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Fourteenth Annual Convention

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VOLUME 16 NUMBER 8

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(INCORPORATING THE AMERICAN JOURNAL OF GASTROENTEROLOGY)

The Pioneer Journal of Gastroenterology, Proctology and Allied Subjects in the United States and Canada

VOLUME 16 AUGUST, 1949 NUMBER 8

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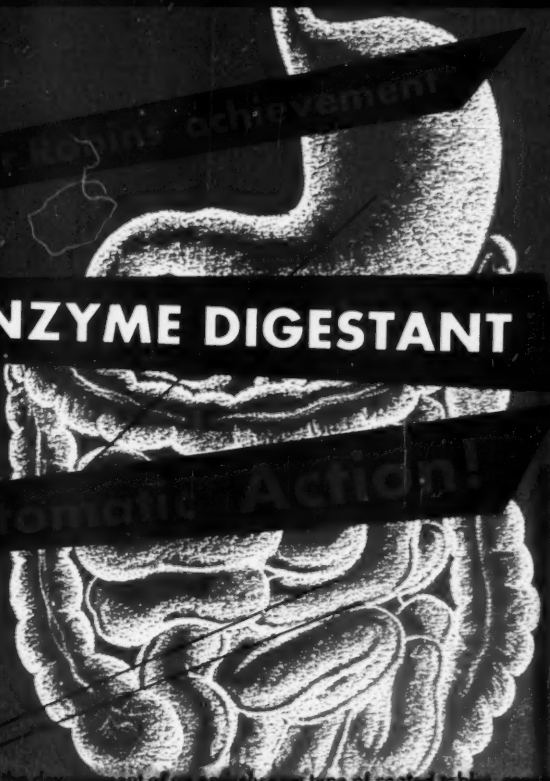

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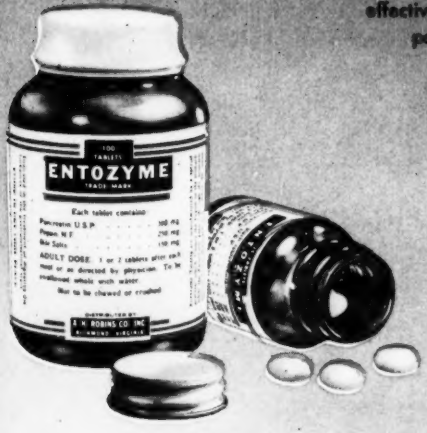

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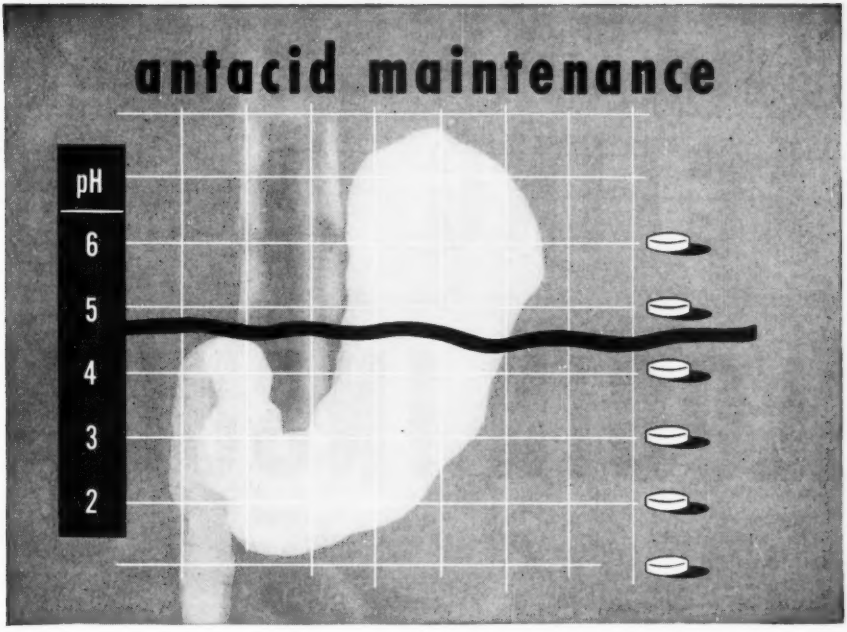

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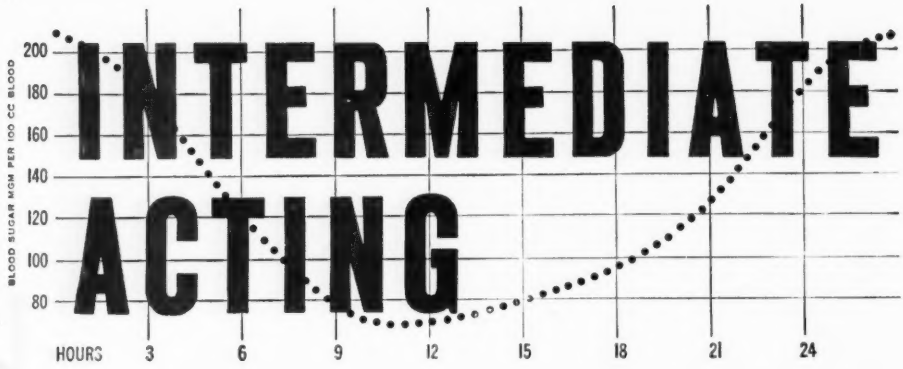

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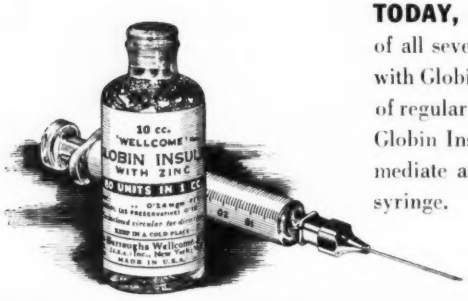

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2. ibid Proc. Am. Diabetes Assn. 8:37, 1948.

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The Review of Gastroenterology

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HISTOPATHOLOGY OF THE SEMISQUAMOUS EPITHELIAL LAYER IN THE GALLBLADDER

FREDERIC DURAN-JORDA*

Manchester, England

It has been described elsewhere that there is a semisquamous epithelial layer covering the whole of the gastric and intestinal mucosae from the cardiac portion of the stomach to the anus, and by developing a special method of fixation, the layer could be studied in detail. This fixative method was applied to the gallbladder and briefly it consists of pinning down part of the organ to be examined in an atmosphere of formaline vapor at room temperature for two or three days. Using this technic, surgically removed gallbladders were investigated, and it was found that this organ, also, is totally covered by a similar semisquamous epithelial layer to that found in the gastric and intestinal mucosae[1, 2].

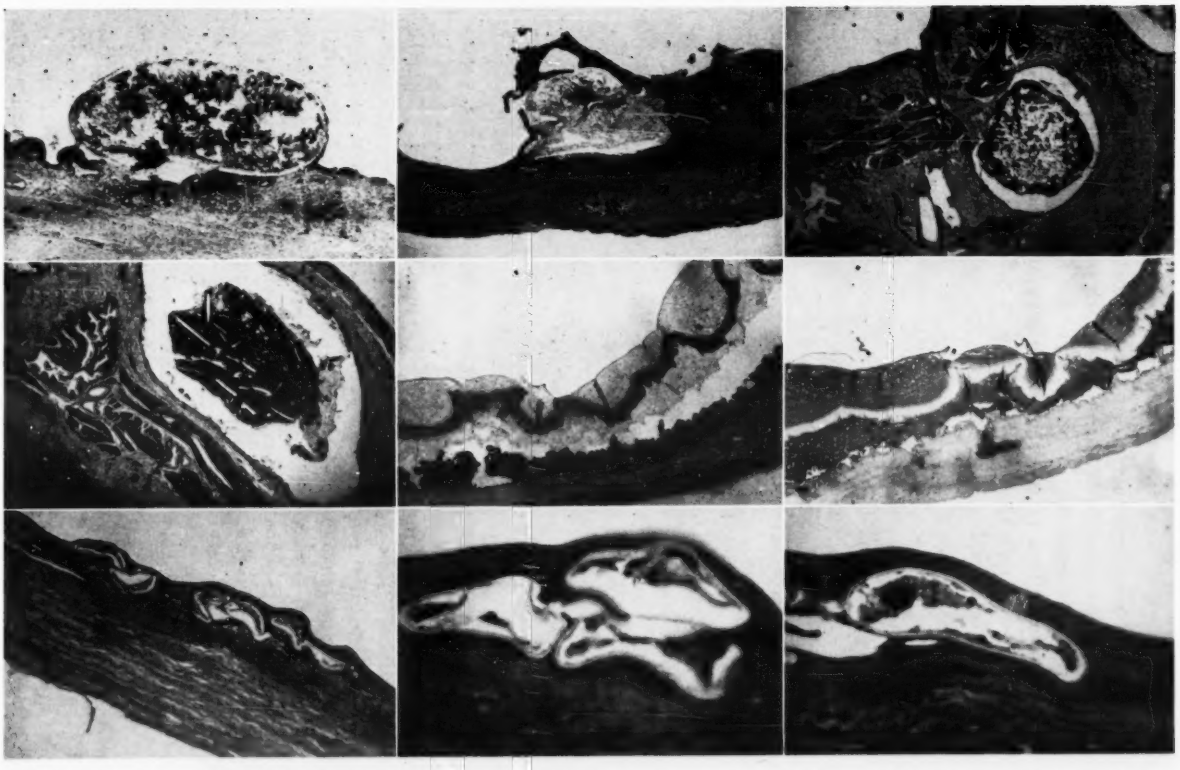
The layer covering the whole of the gallbladder is similar in character to the one described in the small intestine. Its thickness varies from a diameter of one cell to the thickness of two or three cells. In some gallbladders it follows the line of the glands and in others runs along the tops. In some cases, the method of fixation produces a clotting of the fluid contained between the semisquamous epithelial layer and the mucous membrane which makes a cast of the elements. It was also evident that there were very small capillaries present, which, in some pathological conditions are very obvious. When submitted to the action of different mucin stains the layer does not take the stain, but by using the drastic method of microincineration, after which mucus never leaves ashes, it was found that this layer leaves behind a thin deposit of mineral ashes (Figs. 1-5).

Pathology

To study the pathological structure of different gallbladders, use was made of surgically removed specimens. The gallbladder is opened very carefully and its inner surface should not be washed or touched by any instrument. Before submitting it to fixation it is advisable to photograph it or make a schematic drawing of the whole specimen as the process of fixation changes it to a dirty, brown color and some of the smaller lesions fade out. After this the whole gallbladder should be pinned down on to a cork and submitted to formaline vapor fixation (f.v.f.), subsequently cutting out the portions to be studied and embedding them in paraffin blocks, the sections being stained by any of the usual methods.

*Director of Pathology Department, Ancoats Hospital, Manchester, England.

Fig. 1—Upper Left. Normal gallbladder showing a thick semisquamous epithelial layer. Fig. 2—Upper Center. Microincineration of the same section showing the ashes left behind by the semisquamous epithelial layer. Fig. 3—Upper Right. Details of the semisquamous epithelial layer. Fig. 4—Middle Left. Gallbladder of cow. Notice the one cell diameter of the semiquamous epithelial layer. Fig. 5—Middle Center. Human gallbladder, showing capillary included in the semisquamous epithelial layer. Fig. 6—Middle Right. Small magnification of a calculus gallbladder. Notice the atrophy of the mucous membrane but the semiquamous epithelial layer is preserved. Fig. 7—Lower Left. Calculus gallbladder. Mucous membrane has completely disappeared but the semiquamous epithelial layer remains. Fig. 8—Lower Center. High magnification of the bed of a calculus showing the disappearance of the mucous membrane and the presence of the semisquamous epithelial layer. Fig. 9—Lower Right. Inspissated material included in the semisquamous epithelial layer.



Figs. **10, 11**—Upper Left and Center. Small calculus included in the semisquamous epithelial layer. Figs. **12, 13**—Upper Right and Middle Left. Calculi included in the Luschka sinuses of the wall of the gallbladder. Figs. **14, 15**—Middle Center and Right. Dropsy of the gallbladder. Fig. 14 shows a section stained with hematoxylin and eosin which the semisquamous epithelial layer has taken, but the surrounding mucous remains unstained. Fig. 15, stained with mucicarmine the semisquamous epithelial layer does not take the stain. Figs. **16, 17, 18**—Lower Left, Center and Right. Gallbladder of cow. Fig. 16 shows parasites in the semisquamous epithelial layer. Figs. 17, 18 show details of these parasites.

Pseudoulcers

It has been stated elsewhere that in the junction between the stomach and duodenum there are gaps in the mucous membrane which were found to be covered by a healthy semisquamous epithelial layer and were not accompanied by any reaction of the submucosa. This same phenomenon was more noticeable in gallbladders, not only from human specimens but also in different mammals, where some parts of the mucous membrane were missing but the gaps were covered by a very healthy semisquamous epithelial layer which was in contact with the connective tissue. This has not been found to be accompanied by any granulocytic reaction to indicate any inflammatory process. In some gallbladders these gaps were more cellular as a result of hyperplasia of the connective tissue, but after a careful study under high magnification no evidence of inflammatory reaction could be found. The gaps in some parts were only the width of a few cells, and in other places were very appreciable (Figs. 6, 7).

Calculus of the Gallbladder

To study this type of gallbladder, the calculi should not be removed prior to the f.v.f. process but should be left to fall off by the action of gravity during the process. Different observations can be made and atrophy of the mucous membrane can be seen which is probably the result of the pressure of the calculus on the gallbladder wall. The bed of the calculus leaves its shape in the mucous membrane. In some gallbladders, it is interesting to observe that different parts of the semisquamous epithelial layer show the inclusion of some kind of yellowish, inspissated material of variable size and shape. By the accumulation of this material, a small calculus is produced which remains attached to the gallbladder wall and is surrounded by the semisquamous epithelial layer. In another gallbladder, the Luschka sinuses were full of inspissated material, producing a picture of calculosis into the wall of the gallbladder. This phenomenon of retention by the semisquamous epithelial layer suggested that probably one of the mechanisms of stone formation in the gallbladder is the nondialysis of the secreted material through the semisquamous epithelial layer, so that it remains imprisoned in it, grows, and produces a calculus which remains anchored to the gallbladder wall and cannot be expelled by the contractions of the organ. A similar mechanism intervenes in the production of some appendicular fecaliths (Figs. 8-13).

Hydrops of the Gallbladder

One of the gallbladders was very dilated and contained a fair amount of slightly yellow stained fluid. On opening the gallbladder, the material had the appearance of mucus but seemed to have capillaries embedded in it. When the specimen was drained, small bleeding points in the gallbladder wall were found. After f.v.f., embedding and cutting, it was apparent that the thin gallbladder wall was covered by a big deposit of mucus, in the center of which there was organic tissue formed by the semisquamous epithelial layer which contrasted very strongly with the surrounding mucus, and when stained with hematoxylin and eosin showed

that the semisquamous epithelial layer took the eosin very well, leaving the mucus unstained. Using different mucin stains, the semisquamous epithelial layer remained unstained, but the mucus took the color (Figs. 14, 15).

Parasitosis

In the gallbladders of different mammals, especially the cow, parasitosis was present throughout the whole length of the mucous membrane covering the organ, and it could be seen how the parasites were cut at different lengths. Two facts were worthy of mention:— 1) that along the whole length of the organ the parasites built their own burrows into the semisquamous epithelial layer and 2), there was no evidence of any inflammatory cellular reaction. This proved, therefore, that the relation between the parasite and host was a saprophitic one. A similar mechanism of parasitosis in the human appendix has already been described, in which case the burrowing parasite was the oxyuris vermicularis, but in this instance the

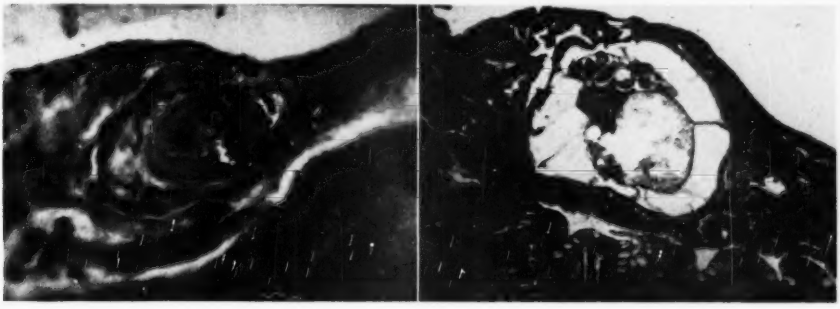


Fig. 19 Fig. 20

Figs. 19, 20—Human appendix showing parasitosis of the semisquamous epithelial layer by the oxyuris vermicularis. Fig. 20 shows a cross section of uterus of the oxyuris vermicularis showing the presence of ova.

semisquamous epithelial layer showed a very extensive cellular reaction, in contrast to the perfectly normal aspect of the mucous membrane and other layers of the appendicular wall (Figs. 16-20).

Cholecystitis Glandularis Proliferans

A case of cholecystitis glandularis proliferans has been studied and it was possible to demonstrate that in spite of the disorganization of the mucous membrane itself, the semisquamous epithelial layer covered the whole of the organ including gaps in the mucous membrane, so producing a picture described as pseudoulcer (Figs. 21, 22).

Carcinoma

We have not been able to study any carcinoma of the gallbladder, nor any adenocanthoma, but it is probable that malignant tumors classified as adenocanthoma may arise from malignant degeneration of the semisquamous epithelial layer and not result from a metaplasia of the mucous membrane cells.

Summary

The author describes the existence of the semisquamous epithelial layer covering and protecting the normal gallbladder, which contains capillaries.

The layer can be studied by microincineration to demonstrate its real organic nature. The author has described the relation between the semisquamous epithelial layer and different pathological syndromes.

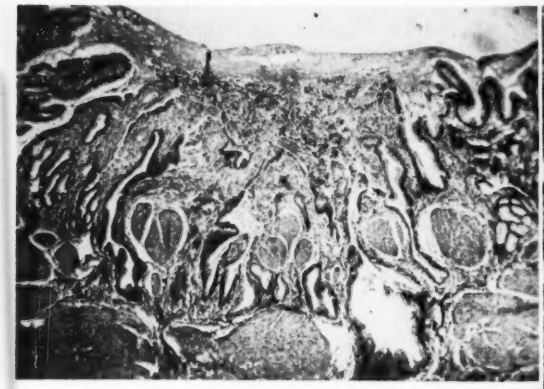


Fig. 21

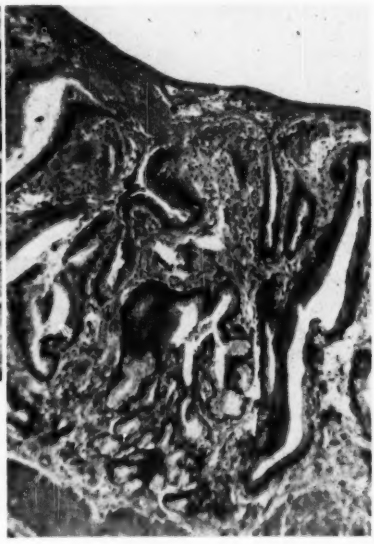


Fig. 22

Figs. 21, 22—Cholecystitis Glandularis Proliferans of the gallbladder. Notice that the semiquamous epithelial layer covers the whole mucous membrane including its gaps.

Acknowledgements

I am greatly indebted to the surgeons of Ancoats Hospital for their kind cooperation in supplying the surgically removed gallbladders used in these investigations, and to Mr. J. B. Dean, B.Sc. for the photomicrographs.

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REGIONAL ENTERITIS IN THE NEGRO*

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and
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It has been stated that regional enteritis does not occur in Negroes or at best is very rare in this race. Both Shaiken[1] and Bockus[2] have cited Crohn that only one case had been reported in a Negro up to 1941. Clark and Dixon[3] in 1939 noted that the disease had not been observed among Negro patients at the Mayo Clinic. White has stated[4] that this disease has rarely been encountered among Negroes and refers to a personal communication from D. C. Elkin that only two cases were recognized at the colored division of Grady Hospital (Atlanta), one at operation, the other at autopsy.

The brilliant description of this disease by Crohn, Ginzburg and Oppenheimer[5] appearing in 1932 involved fourteen cases, all Jewish patients. Subsequent reports continued to stress the greater incidence of cases in this race. However, as the American and world literature on this condition expanded, numerous reports disclosed its existence in almost all countries and races. Crohn[6] has commented on its widespread geographic distribution and infers that it affects all races, including the Negro and Mongolian.

In view of this universal dissemination, it seemed profitable to investigate its occurrence in Negroes by a review of the literature and of the cases seen at the Harlem Hospital, New York which has an average of 20,000 admissions yearly, the preponderant number of which are Negro.

Review of Literature

A review of 299 papers appearing in the American (i.e. U.S.) literature on the subject since 1932 (the date of the original publication by Crohn, Ginzburg and Oppenheimer) was made. Because considerable variation in terminology exists, all articles that appeared to have any bearing on this subject were consulted. This included therefore, reports on terminal or distal ileitis, segmental ileitis or enteritis, ileocolitis, cicatrizing ileitis, hypertrophic stenosing enteritis, interstitial enteritis, etc., etc.

Only twenty-six cases were found in Negroes, although it must be admitted that in many of the reports no mention of race is made. These are summarized in Table I. Three additional cases reported in Negroes are not included because they showed no small bowel involvement and the description of the colon pathology was too vague to be of value.

---

*From the Medical Department of Harlem Hospital (Department of Hospitals, New York City) Dr. Barnet P. Stivelman, Director.
†Visiting Physician, Harlem Hospital; Associate Attending Physician, University Hospital; Attending Gastroenterologist, Beekman-Downtown Hospital, New York City.
††Assistant Visiting Physician, Harlem Hospital.

Despite this relatively small number of cases, several presented numerous interesting features. In seven cases the preoperative diagnosis was appendicitis—emphasizing the difficulty of distinguishing the acute phase of regional enteritis from appendicitis. Cutler's[11] case showed an obstruction by barium enema in the mid-transverse colon and a preoperative diagnosis of obstructive carcinoma was made. At operation an inflammatory lesion was found. Ten months later when the patient was reoperated it was found that the disease now involved the

TABLE I

Author	Date	No. of Cases	Age	Sex
Meyer and Rossi[7]	1936	1	34	M
Sanders[8]	1936	2	59 30	M M
Grimes and Massie[9]	1938	1	23	M
Stafford[10]	1938	4	—	—
Cutler[11]	1939	1	47	F
Lehman[12]	1939	1	14	M
Browne and McHardy[13]	1940	1	21	M
Eckel and Ogilvie[14]	1941	4	31 29 26 14	F F M M
Meade[15]	1941	1	50	M
Smith[16]	1941	1	48	F
White[4]	1941	2	—	—
Wilson, Grinnan, Ashburn[17]	1942	1	21	F
Smithy[18]	1943	3	23 21 27	M M F
Johnson[19]	1943	1	58	M
Bockus[20]	1945	1	38	F
Rees[21]	1945	1	32	F

cecum, appendix and three centimeters of terminal ileum as well as the sigmoid. Browne and McHardy[13] discuss the relationship to trauma and present a case which, seven days after severe trauma to the abdomen, complained of cramping pain in the left lower quadrant, relieved by vomiting. Mild diarrhea, melena and low grade fever were also present. X-rays showed distention of the proximal portion of the jejunum. A resection was performed and the gross pathology revealed six inches of jejunum with an acutely congested serosal surface. The thickened leathery wall showed acute exudate and chronic inflammatory changes. The lumen

was constricted to 4 mm. Two other cases in which the lesion was limited to the jejunum have been reported. One of these[19] showed multiple napkin ring constrictions of the jejunum, the highest being three inches below the reflection of jejunum at the muscle of Treitz, the next was five inches distal with the intervening bowel hugely dilated and at 6″ to 11″ intervals five additional napkin ring lesions were found in the upper jejunum. The remainder of the small bowel was normal. Rees[21] reported a case in which the disease was limited to the proximal jejunum and showed complete obstruction at three isolated places.

Case Reports

To these twenty-six cases, we should like to add four cases seen at the Harlem Hospital between 1936 and 1948.

Case 1:—L. H., Male, Age 49. This Negro patient entered July 9, 1939 with a history of chills and fever which began on July 6th and subsided with self-medication. The following day he complained of cramps in the lower abdomen, moving from side to side. No relief was obtained after taking bicarbonate of soda, epsom salts and an herb medicine. There was no nausea or vomiting and the bowel movements were normal. The history was negative as to any respiratory or urinary symptomatology. Past history revealed treatment for syphillis and gonorrhea ten years previously.

Physical examination revealed an obese, well developed male, lying comfortably in bed. The essential findings were limited to the abdomen, with tenderness on deep palpation in the right lower quadrant and to a lesser degree in the left lower quadrant. Rebound tenderness in the right lower quadrant was equivocal. The temperature was 99°; the white blood count was 12,500 with 76 per cent polymorphonuclear and 24 per cent lymphocytes; the urine showed a trace of sugar.

Laboratory tests revealed a negative Kahn, creatinine 1.3 mg. per cent, urea nitrogen, 13 mg. per cent, and blood sugar 130 mg. per cent.

The patient was operated on July 10th with a preoperative diagnosis of appendicitis. The appendix was found to be normal but the distal four inches of ileum showed marked bluish discoloration and a thickened wall covered with exudate. The mesentery of this portion of small intestine was also thickened and indurated. There was no free fluid in the abdomen. The portion of avital ileum was resected and the appendix removed. The postoperative course was stormy. Glycosuria was present for three days, reaching 4 plus on one occasion, but this was controlled by small doses of regular insulin. The temperature on the first postoperative day was 102.6 with a pulse of 120 and respirations of 28 per minute. The lungs revealed coarse rales on both sides. On the second postoperative day, the patient was restless, breathing more rapidly, and the abdomen was distended and moderately tender. There was some dullness at the left base together with coarse rhonchi. It was felt at the time, that the signs were due to distention rather than any intrinsic lung pathology. A bedside x-ray of the chest was negative. On the morning of the third postoperative day the patient was more comfortable. That

afternoon the lungs showed dullness at the left base and bronchial breathing; the abdomen was distended and there was a moderate amount of drainage of a foul odor. The patient continued to go downhill and despite a transfusion of 500 cc. whole blood and supportive therapy, he died on the fifth postoperative day.

No autopsy was obtained. The pathology report on the surgical specimen was terminal ileitis and normal appendix.

Case 2:—J. H., Male, Age 38. This Negro patient was admitted August 24, 1944 with a history of dull pain around the umbilicus which started August 21st. The next day the pain had shifted to the right lower quadrant where it remained until the time of admission. There was no vomiting and the bowels were regular. He had felt feverish on August 22nd but had no chills. There was slight anorexia. This was the first such attack. Past history revealed antiluetic therapy had been started one year ago and the patient was receiving treatment until time of admission.

The temperature was 101.8, pulse 86, respirations 12. The essential findings were limited to the abdomen where tenderness on palpation and rebound tenderness were noted in the right lower quadrant. The rectal examination revealed tenderness to palpation on the right. The white blood count was 14,900 cells of which 85 per cent were polymorphonuclears and 15 per cent lymphocytes.

The diagnosis was acute appendicitis and the abdomen was opened through a McBurney incision. A normal retrocecal appendix was found but the cecum and terminal ileum were plastered with organized adhesions, the serosa was reddened, and the terminal six inches of ileum were reddened, thickened and indurated. The original incision was closed and the abdomen was reopened through a left upper rectus incision. The ileum was transected 10" from the ileocecal valve and a side-to-side ileotransverse colostomy was done.

The microscopic diagnosis of the specimen was ileum showing edema and chronic inflammation.

The postoperative course was uneventful. A gastrointestinal series done on September 9, 1944 showed no lesion of the gastrointestinal tract. The patient was discharged on the twenty-first postoperative day.

Case 3:—D. B., Male, Age 29. This Negro patient was admitted August 11, 1945 with a two week history of diarrhea. Preceding the onset of diarrhea, he had complained of indefinite abdominal pain which could not be localized. He was having three to five bowel movements a day. The stools were watery and odorous. He had lost fifteen pounds in the previous three months. He had been under his physician's care, but showed no improvement, and was referred to the hospital.

Physical examination revealed a poorly nourished acutely ill male. The essential findings were limited to the abdomen which was scaphoid in shape. There was no tenderness. The liver, spleen and kidneys were not palpable. In the region of the cecum a mass was felt which was firm and nonmovable. He was afebrile. Laboratory data were normal. The patient responded to supportive

therapy and four days after admission was asymptomatic. A barium enema showed deformity of the terminal ileum suggesting regional ileitis.

The patient was discharged August 22, 1945 to the gastrointestinal clinic for further observation.

Case 4:—J. F., Male, Age 39. This Negro patient was admitted October 9, 1946 with a history of periumbilical pain of three days duration. He had nausea with frequent vomiting since the onset of his illness. Bowel movements had been regular until two days before admission and there had been no evidence of bloody diarrhea. Pain was sharp and intermittent. Past history revealed asthma of many years duration, and a similar episode of abdominal pain one year ago. The physical examination disclosed an acutely ill male. The heart was negative and the breath sounds were consistent with the history of asthma. The abdomen was flat, liver and spleen were not enlarged, nor were there any palpable masses; peristalsis was present; there was generalized tenderness, direct and rebound, and rigidity. Rectal examination was negative. The white blood count was 12,500 cells with 75 per cent polymorphonuclears, 15 per cent transitional cells, and 10 per cent lymphocytes. The temperature was 98.6, pulse 80.

The clinical diagnosis of acute appendicitis with possible peritonitis was made.

At operation the appendix was found to be somewhat injected but not acutely inflamed. Two to three hundred cc. of serous fluid were found in the peritoneal cavity. The terminal ten to twelve inches of ileum were pale, and thickened to about four times normal size and the mesenteric nodes were enlarged. Appendectomy was not done.

The culture of peritoneal fluid was sterile. The postoperative course was uneventful and the patient was discharged October 24, 1946.

Discussion

Since many of the published papers fail to mention the race of the patient, more than twenty-six cases of regional enteritis in the Negro may exist. However, we must conclude both from the literature and our own experience that the disease is rare in this race, although we can offer no explanation for this observation. The findings do not agree with the observations of Bockus[2] who, noting the absence of the disease in families in the upper economic brackets, considered a factor of subnutrition as a possible etiology. We cannot help but feel that if subnutrition played any significant role, the incidence in the Negro should be much higher. Further, climate has been said to play some part in this disease (Bockus[2]); no cases having been reported from Latin America. While the number of cases reported has been relatively few, we found no significant difference between Northern and Southern centers in the United States. The Central and South American literature was not reviewed but in a recent article, Ascencio-Camacho[22] claims that acute segmental enteritis is a frequent disease among native-born Puerto Ricans. We have seen one case in a 45 year old Puerto Rican female.

We have no personal experience with the casual relationship of trauma. Browne and McHardy's[13] case is provocative. They cite other cases in the literature, though not in Negroes, where trauma was considered an etiological factor. In symptomatology, surgical aspects and pathology, the lesions were those of regional enteritis.

As far as can be determined, cases in the Negro do not differ in the sex distribution of three males to two females nor from the average age incidence which is about thirty years.

No case of the fistulous stage of this disease has been reported in the Negro nor are there any familial cases recorded in this race.

Summary and Conclusions

1. Twenty-six cases of regional enteritis in the Negro have been collected from the American literature since 1932.

2. Four additional cases are reported.

3. Regional enteritis is a rare disease in the Negro.

(We wish to thank Dr. Louis T. Wright, Director of the Surgical Service, Harlem Hospital for permission to report some of these cases).

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GALLBLADDER DISEASE IN THE AGED*

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In preparing the subject matter for this presentation, the question naturally arises, "Does gallbladder disease in the aged patient differ from gallbladder disease in the middle aged person or the young adult?". The answer becomes at once apparent that the symptomatology in the uncomplicated aged gallbladder patient in no way differs from the manifestations displayed by gallbladder patients in other age groups. The exception is that the process of senescence with its inevitable physiologic alterations, does render certain sequelae, complications and associated diseases more common in the aged gallbladder patient, and the recognition and proper management of some of those conditions may at times tax our abilities to the utmost. I shall return to the discussion of these later on in more detail.

As to the frequency of gallbladder disease in the aged, it is common knowledge that the incidence of cholelithiasis steadily increased with age. In an interesting survey conducted by Blumberg and Zisserman[1] they found gallstones in 70 per cent of all patients over seventy years of age. Although the statistical results of similar surveys may differ somewhat, this is not sufficient to be of any practical value. There is of course considerable evidence to support the view that various alterations of a metabolic or endocrine nature as well as newly acquired sedentary habits of the aged, not infrequently created by disease in other systems, are all responsible etiologic factors insofar as disease of the biliary tract is concerned.

It would be appropriate at this juncture to review briefly the most recent knowledge of the etiology and pathogenesis of gallbladder disease so as to gain a clearer appreciation of the role these play in the production of biliary tract disease generally, and how these factors may become accentuated in the older patient.

Up to about two decades ago the role of infection was accorded primary importance in the causation of gallbladder disease. Great stress was placed upon the frequent elicitation of a history of typhoid fever from patients harboring gallstones. Undoubtedly the incidence of a history of typhoid was much more frequent years ago than it is now, when typhoid fever is rarely seen, especially in our larger cities. Nevertheless, in spite of the rarity of typhoid, the incidence of gallbladder disease has not diminished. Some investigators have reported the recovery of pathogenic organisms, such as the streptococcus or B. Coli in from 10 to 66 per cent of gallbladders containing gallstones. On the other hand, it is the contention of many authoritative investigators that the finding of living

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bacteria in gallstones does not prove their primary responsibility for the stones nor for a preexisting cholecystitis, since in a large percentage of cases of chronic cholecystitis, with or without cholelithiasis no evidence of any infecting organism is found. Bockus and his coworkers[2] at the Pennsylvania Graduate Hospital believe that the colon bacillus and probably the streptococcus may be recovered from the bile by biliary drainage when they are really in transit from the liver. The liver, as is well known may receive these organisms from the intestines via the portal circulation and excrete them with the bile into the gallbladder and eventually again into the intestine, where they are finally disposed of. In this connection it is also important to call your attention to the fact that bile itself is a great deterrent to the growth of bacteria. If the concentration of bile salts in the gallbladder is 70 per cent of normal or more, no growth of bacteria will occur. In fact the growth of organisms in bile has been shown to occur in inverse proportion to the concentration of bile salts.

This brings us to the consideration of other factors which are at present considered as the most probable ones in the etiology of gallbladder disease, namely mechanical, chemical, metabolic and in some instances, endocrine. It has been amply demonstrated both in the animal and human that the proper concentration of bile salts in the gallbladder bile not only acts as a deterrent to infection but also maintains the bile cholesterol in solution, thereby preventing its precipitation within the gallbladder, thus forming a nucleus of cholesterol crystals which adhere to the gallbladder mucosa and act as a nidus for the formation of gallstones. An adequate amount of bile fatty acids also aids in keeping cholesterol in solution. It becomes readily discernible then, that a delicate chemical balance exists between the bile cholesterol and bile salts or acids. Any mechanism which tends to disturb this balance either in the direction of diminished bile salts or excess of cholesterol, will initiate the process of gallstone formation, with subsequent trauma to the gallbladder mucosa and in some cases, ultimate superimposed infection of the gallbladder mucosa. It is the contention of the majority of investigators in this field that infection when it does occur, is secondary to trauma. The precipitation of calcium from bile was also shown to be not infrequently associated with cholesterol precipitation, especially in acute obstructions or chronic repeated obstructions of the cystic duct or the neck of the gallbladder.

The most important, if not the most common mechanism which initiates this pathophysiologic process, is stasis within the biliary apparatus, and particularly stasis within the gallbladder. The latter may be caused by any one, or a combination of factors. In the young adult female, pregnancy is an outstanding example of gallbladder stasis, especially in the later months. Tight corsetting has been given as another cause. Colonic stasis, particularly cecal stasis, as has been shown by Ivy, favors gallbladder stasis. Low fat intake and sedentary habits are also important factors. We can readily see what important roles the last three factors can play in the production of stasis and initiation of gallbladder disease in the aged. Of course gallbladder stasis may also be produced by various inflamma-

tory or anomalous conditions involving the extrahepatic, and particularly the cystic duct and the wall of the gallbladder itself.

As to the role of metabolic and endocrine factors, in the genesis of gallstones and gallbladder disease, I might call your attention to the relationship of the thyroid gland to cholesterol metabolism. The association of hypothyroidism, which is not uncommon in the aged, with hypercholesterolemia is well known. It is possible, though not definitely proven, that in some cases a pituitary insufficiency may be responsible for the hypothyroid state in the aged, just as it causes a depression of the adrenocortical or ovarian function. The effect of a hyperparathyroidism in the production of a hypercalcemia and gallstones is well known and serves as another example of an endocrine-metabolic disturbance affecting the biliary tract. Hypercholesterolemia and cholelithiasis are commonly associated with diabetes, especially in the aged patient, and this association again tends to emphasize the interrelationship between a metabolic-endocrine disease and gallbladder disease. The inevitable process of senescence with its associated degenerative changes, tends to hasten and accentuate these existing pathological processes.

It would constitute a grave error of omission were I not to mention the role of vitamins in gastrointestinal and particularly in gallbladder disease; especially since avitaminosis or subvitaminosis is such a common finding in the geriatric patient. Rothman[3] has postulated an indirect relationship between Vitamin C and gallstone formation and Saiki[4] was able to produce gallstones in animals by feeding them diets deficient in Vitamin A. Whether these findings have any definite clinical implications or applications, is not certain though possible.

In discussing the symptomatology of gallbladder disease, I need not go into great detail at this time, since you are all familiar with the type of patient who for many years, either continuously or intermittently presents a symptom complex of upper abdominal distress usually appearing shortly after meals, associated with abdominal bloating, belching, pyrosis and sour eructations; a history of intolerance to fats associated with some right upper quadrant pain and tenderness on palpation; or the patient who gives a history of repeated attacks of biliary colic, with or without bouts of jaundice, the latter frequently due to concomittant choledochal obstruction. In the aged patient, and particularly one who has harbored gallbladder disease for three or four decades, the accompanying morbidity is much greater and an ever-existing potential source for certain sequelae and complications. These may manifest their appearance in either a carcinoma of the gallbladder or common duct; or in bouts of acute cholecystitis sometimes eventuating in a slow or an acute perforation of the gallbladder with a resultant formation of an internal biliary fistula, either into the small intestine or colon. Obstruction of the bowel as a result of a gallstone impaction or a calculous obstruction of the common duct are not too infrequent. Any one of the aforementioned conditions poses a difficult and serious therapeutic problem, especially in the aged.

Associated disease in the gastrointestinal tract is not infrequently encountered in the geriatric patient, such as a hiatus hernia or an epigastric hernia, and these

must be thought of in our diagnosis and seriously considered in the differential diagnosis, as being the possible cause of the symptomatology in a patient known also to have gallbladder disease.

The disturbances in the cardiovascular-renal systems in the older age group are well known. In this connection it might be interesting to present briefly the results of a statistical survey made by Glenn[5] of the Department of Surgery of the N. Y. Hospital on a series of 276 gallbladder cases in patients over the age of fifty. The incidence of systemic disturbance was as follows: Hypertension—45 per cent; arteriosclerosis—50 per cent; cardiac disease—40 per cent; renal disease—8 per cent and diabetes—10 per cent. As a result of this study the author concludes that the incidence of organic disorders and in particular those of the cardiovascular system, is higher in patients with, than in those without biliary tract disease. In other words, biliary tract disease seems to be conducive to degenerative changes in the cardiovascular system. Indeed gallbladder patients, especially in the older group, not infrequently present themselves with pain and tenderness in the right upper quadrant, disclosing upon examination a large tender liver, as a result of a congestive heart failure. The diagnosis of acute cholecystitis in a patient of this type, known to have gallbladder disease, may be erroneously entertained and digitalis and mercurials withheld at a time when they would most serve their purpose.

Likewise the pain of coronary occlusion may at times be confused with that of cholelithiasis or vice versa, especially posterior coronary occlusions and those myocardial infarctions which are initiated with a good deal of reflex vomiting. The latter is quite common in the older patient. At times, positive electrocardiographic findings following a severe attack of biliary colic may be very confusing; and conversely, in an acute coronary occlusion one may find no electrocardiographic findings for a week or ten days following an attack. These exceptions, which must be borne in mind are encountered in the older patient and require close and repeated observations as well as sound clinical judgement. It is interesting here to recall the fact that Ravdin and his coworkers[6] have, a number of years ago, shown that a gallbladder or common duct and cardiac reflex pathway exists. Distention of either the gallbladder or the common duct, can decrease the coronary flow and alter the cardiac rhythm and rate. The abnormal reflex was furthermore shown to be more sensitive in cases with preexisting cardiovascular abnormality, such as we expect to find in high percentage of our older patients.

Medical management of gallbladder disease in the aged does not materially differ from that of the younger patient. With a good many of these patients dietotherapy is no particular hardship, since most of them have lived on a restricted diet for years and have learned through experience to be very discrete. A bland, moderately high protein and carbohydrate diet of low fat content, is most suitable for them. This should be supplemented with daily vitamins, either orally or parenterally. I would like to sound a word of caution regarding vitamins; some gallbladder patients do not tolerate preparations containing cod liver oil or halibut oil. It is advisable not to prescribe these, if they cause even minor di-

gestive upsets. The diet of a diabetic patient with gallbladder disease must of course be modified in accordance with the severity of the condition, and proper control maintained, usually with the aid of insulin. Proper bowel hygiene must be maintained, frequently with the aid of a mild laxative, since atonic constipation is a common condition in the older patient.

Choleretics in 6-8 week courses, two to three times a year is a sound procedure in the conservative management of the gallbladder patient. These may be used either with or without antispasmodics and mild sedatives, in accordance with the requirements of the individual case.

An acute gallbladder emergency in the geriatric patient may at times precipitate some degree of cardiac failure, and proper therapeutic measures for the latter complication will help to tide the patient over, until the acute episode is under control. Antibiotics may prove necessary and even life-saving in some cases complicated by an infectious process. Parenteral therapy may have to be resorted to in some cases for a longer or shorter period of time; and here again, one must be very cautious not to overload the heart and precipitate a pulmonary edema. We must remember that the cardiovascular system grows less resilient with age. Smaller quantities of intravenous fluids, given at slower rates will help to prevent such accidents. At times, fluids by hypodermoclysis only, may be advisable.

Obviously it is unwise to expose the aged person with advanced arteriosclerotic changes to operation for uncomplicated cholelithiasis if they can be maintained relatively comfortable by conservative therapy. Nevertheless many aged patients have been successfully operated on for cholelithiasis, particularly when complications such as acute cholecystitis or persistent jaundice, have made surgical intervention mandatory. We have learned from experience that the mere factor of age is not a contraindication to surgery. As Rowntree[7] has said, the aged are very often "good livers and take a lot of killing". Furthermore, the marked improvement in the preoperative preparation and postoperative care, as well as in the technic of gallbladder surgery, in the past two decades, have greatly reduced the hazards and mortality of biliary tract surgery in the aged patient. In a report by Quigley[8] on 100 consecutive cases over sixty-five years of age operated on for acute gallbladder disease, there was an overall mortality of only 13 per cent. This is an excellent record considering the age of the patient and the nature of the condition requiring emergency surgical intervention.

In conclusion I wish to state, that I have attempted in a brief manner to touch upon the salient features concerning gallbladder disease in the aged. I have particularly tried to stress some of the sequelae, complications and associated diseases as they may manifest themselves in the geriatric patient. While stressing the satisfactory results of the conservative and intelligent management of the majority of patients with gallbladder disease, I also wanted to emphasize the fact that if and when necessary, surgical intervention is seldom contraindicated because of age. The increasing proportion of the aged in the general population has made us more aware of the various geriatric problems confronting us in general, and because of its frequency, gallbladder disease in particular.

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TRAUMATIC DIAPHRAGMATIC HERNIA WITH GASTRIC OBSTRUCTION AND JAUNDICE*

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Hernias through the diaphragm may be of three types—the congenital, the acquired and the traumatic. In this paper we propose to deal with the traumatic type.

In a comprehensive review of the literature, we have been unable to find any case of traumatic diaphragmatic hernia complicated by gastric obstruction and jaundice. We therefore present our case report as being, to the best of our knowledge, the only one of its kind on record.

Case Report

A 25 yr. old male, Puerto Rican, longshoreman, was admitted to the Cumberland Hospital on August 7, 1946, for severe epigastric pains and vomiting.

His present illness began about 6 months prior to admission with the onset of occasional bouts of epigastric pains and vomiting. The vomitus was at times of the "coffee grounds" variety. The pains came on about ½ hour after meals, sharp, colicky, with no radiation and relieved by vomiting. About one month before admission to the hospital the patient noticed black stools on various occasions. These symptoms became progressively worse. There was a history of irregular dietary habits and indulgence in alcoholics.

Past history:—Nine months before admission to the hospital the patient had received a stab wound in the left anterior chest wall.

Upon examination the patient appeared to be acutely ill. There was a healed scar about ½" long in the left anterior axillary region. The respiratory and cardiovascular systems were essentially normal. Abdominal examination disclosed tenderness in the epigastrium.

A tentative diagnosis of bleeding peptic ulcer with pyloric obstruction was entertained and appropriate therapy instituted.

The patient's condition became worse, with the appearance of dyspnea, rapid feeble pulse and jaundice.

Examination of the chest at this time revealed prominence of the left hemithorax with flatness on percussion and absent breath sounds on auscultation. The point of maximal cardiac impulse was to the right of the sternum, which indicated a shift of the mediastinal structures to the right.

In view of the altered clinical picture and the history of a previous stab wound in the left chest, the diagnosis of traumatic diaphragmatic hernia with

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gastric obstruction was considered. The jaundice was explained on the basis of torsion and edema of the second portion of the duodenum, including the ampulla of Vater, thus giving rise to extrahepatic obstructive icterus.

Laboratory findings revealed:—

1. Hemoconcentration as a result of persistent vomiting.
2. Urea nitrogen was elevated.
3. The icteric index was 49 units.
4. Liver function tests were negative.

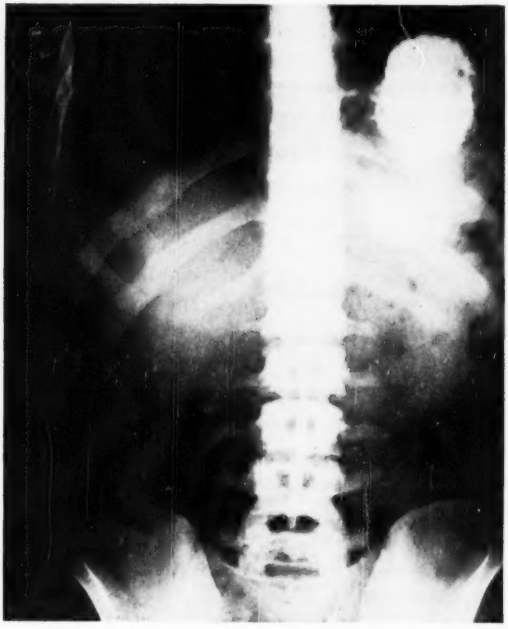

Fig. 1—Roentgenogram showing a biloculated stomach.

X-ray of the chest on August 12, 1946, revealed the heart shifted to the right of the sternum. The stomach was tremendously dilated, with the fundus noted at the second rib anteriorly. A diagnosis of possible eventration was considered by the roentgenologist.

A radiographic study of the gastrointestinal tract on August 14, 1946, revealed a biloculated stomach with the pyloric portion higher than the fundus (Figs. 1, 2). A 12 hour film did not show any progress of the barium beyond the herniated portion of the stomach. The patient was transferred to surgery on August 16, 1946. At first a left phrenocotomy was performed. On August 24, 1946, repair of the herniation was done. Upon entering the peritoneal cavity a rent approximately 3"

in diameter was found in the left lateral leaf of the diaphragm, midway between the dome and the costophrenic sinus. Jammed into the opening was about ¾ of the stomach, including the pars media and pyloric region, as well as a large portion of the transverse colon. The constricted portion of the stomach was edematous and fibrotic. The liver was normal. The gallbladder, although distended, could be compressed easily.

The contents of the hernial sac were reduced and the tear in the diaphragm was repaired. The patient made an uneventful recovery and was discharged from the hospital on September 8, 1946.

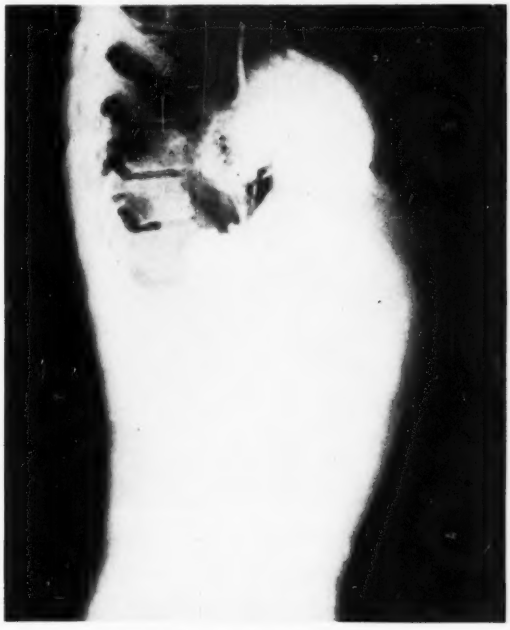

Fig. 2—Shows the stomach with the pyloric portion higher than the fundus.

A roentgenologic study of the gastrointestinal tract on October 15, 1947, was negative for pathology (Fig. 3) and the patient was clinically well.

Discussion

Much attention has been directed in recent years to diaphragmatic hernia, particularly to the congenital and nontraumatic acquired forms. Less consideration has been given to those of traumatic origin developing or producing symptoms at a period more or less remote from the time of the original injury. Every case of left sided gunshot or stab wound of the chest or crushing accident of the trunk may be one of potential diaphragmatic hernia.

Nasau[1] reported a case where 20 months elapsed from the time of the accident until the condition of the traumatic diaphragmatic hernia was disclosed by x-ray examination.

Polson[2] described a case in whom the diagnosis of diaphragmatic hernia was made on autopsy 12½ years after a gunshot wound. The occurrence of symtoms of acute abdomen in the presence of evidence of tabes dorsalis pointed to a gastric crisis.

Mast and McDonough[3] reported a case of traumatic diaphragmatic hernia resulting in intestinal obstruction. The hernia occurred 8 months following the initial injury to the diaphragm by a gunshot wound to the upper abdomen and lower left thorax.

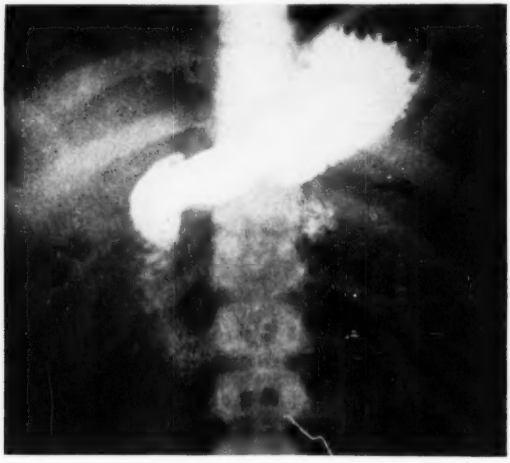

Fig. 3—Roentgenogram showing a normal stomach 14 months after operation.

Boeck and Cook[4] published a case of an unusual right diaphragmatic hernia where the condition remained undiagnosed for almost 7 years after an automobile accident.

In our patient the diagnosis was made 9 months after a stab wound in the left anterior chest with injury to the diaphragm.

With improved methods of diagnosis, many diaphragmatic hernias are being discovered where formerly they were not recognized until they reached the operating table or at autopsy.

The symptomatology in cases of large diaphragmatic hernias has been discussed under 3 headings by Morein[5].

(1) Impairment of function of herniated viscera may account for a group of complaints. The character of the symptoms depend upon the size of the hernia, the viscus which have herniated, and whether or not the herniated structures are fixed or slide back and forth between the abdomen and thorax.

(2) Impairment of function of the thoracic structures as a result of pressure from the herniated organs accounts for another group of symptoms.

(3) Impairment of diaphragmatic function occurs in some cases.

Our patient presented abdominal as well as thoracic symptoms and signs which are usually described in the literature and in addition the sign of jaundice.

The diagnosis of traumatic diaphragmatic hernia can be based upon symptoms, clinical signs, and a history of gunshot or stab wound of the chest or crushing accident of the trunk. Roentgenological examination should confirm the diagnosis.

Summary and Conclusion

A case of traumatic diaphragmatic hernia diagnosed intravietam and before operation is reported.

In addition to abdominal and thoracic symptoms and signs this case developed extrahepatic icterus. To our knowledge traumatic diaphragmatic hernia complicated by jaundice has not been reported in the literature.

How the jaundice occurred was a matter of pure conjecture. The clinical impression was that if the pyloric portion of the stomach entered the thorax it would result in torsion and edema of the second portion of the duodenum including the ampulla of Vater, giving rise to the picture of extrahepatic obstructive jaundice. This was proven at operation.

The earlier the diagnosis, the better the prognosis.

There is no better treatment than the surgical operation.

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THE USE OF ULTRAVIOLET BLOOD IRRADIATION IN TYPHOID FEVER*

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and
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This preliminary report is intended to compare cases of typhoid fever, admitted to Shadyside Hospital for a ten year period beginning May, 1938, as to mortality and morbidity, with and without, the use of ultraviolet blood irradiation therapy (Knott technic). The cases were treated by various Staff physicians, and the Department of Blood Irradiation did nothing but administer blood irradiation therapy. These cases were diagnosed by: (1) clinical histories; (2) blood counts; (3) Widal agglutination tests or the recovery of the typhoid bacillus in either blood, stool or urine or combinations (See Table I). There were six cases out of a total of nine treated with ultraviolet blood irradiation therapy with no deaths, three of them by the combination of irradiation therapy and sulfonamides and three with irradiation therapy alone. Three cases were not irradiated in the series, with one death.

CASES IRRADIATED	CASES NOT IRRADIATED
Case 1. #95136 Miss K., age 5.	Case A. #79508 Mr. M., age 33.
Case 2. #97534 Miss S., age 19.	Case B. #108799 Miss G., age 25.
Case 3. #111672 Miss R., age 19.	Case C. #120885 Mrs. C., age 47.
Case 4. #86273 Mrs. L., age 30.	
Case 5. #86350 Miss L., age 7.	
Case 6. #88182 Mrs. B., age 34.	

CASES IRRADIATED

Case 1:—Miss K., age 5, was admitted 10/26/41, with a four day history of general malaise, frequency of urination with nocturia and intermittent high temperature. On admission her temperature was 103.2, pulse 140 and respirations 30. Admission blood work revealed 3,780,000 RBC., 9,500 WBC., 77 per cent neutrophiles, 19 per cent lymphocytes, 4 per cent monocytes. Widal agglutination was positive in "O" antigen (1:320) and "H" antigen (1:180), and on repeat both were positive (1:320). Blood culture on the second hospital day revealed E. Typhosis, and the stool culture on the 8th hospital day was positive for E. Typhosis. Urinalysis on admission revealed a positive reaction for acetone and diacetic acid but was negative for E. Typhosis. Patient received sulfonamide therapy for two days after admission, totaling 64¼ grains. UBI therapy was given on the third and fourth hospital days. The patient was discharged on the 13th hospital day despite the fact that she was still running a typical typhoid temperature curve. Her family physician, for reasons pertaining to her home, signed her out with the notation, "Patient's condition improving; apparently all toxic symptoms over. To go home to bed isolation under my care." She made an uneventful

*Read before The American Blood Irradiation Society, Chicago, Illinois, June 20, 1948.
†Department of Blood Irradiation, Shadyside Hospital, Pittsburgh, Pennsylvania.

recovery at home. Temperature was normal two weeks (31st day of illness) following discharge. Five stool examinations were negative when checked by the Public Health Department between the 31st and 38th day of illness.

Case 2:—Miss S., age 19, was admitted 2/19/42, with a one week history of general malaise, weakness, loss of appetite, severe vomiting with blood, cough, epistaxis and marked diarrhea occurring every one-half hour. Admission temperature was 103.6, pulse 124 and respirations 28. Admission blood work revealed

TABLE I

DIAGNOSTIC CRITERIA FOR TYPHOID FEVER

Case	1	2	3	4	5	6	A	B	C
History of Exposure					√	√	√		
Temperature	√	√	√	√	√	√	√	√	√
Pulse (Typhoid)			√			√		√	
Headache				√	√	√	√		
Abdominal Tenderness			√	√	√	√		√	√
Diarrhea		√	√					√	√
Epistaxis		√			√				
Cough		√					√		
Mental Dullness	√	√				√	√	√	√
Rose Rash									
Splenomegalia						√			
Leucopenia	√	√	√	√	√	√	√		
Widal Agglutination	√	√	√	√		√	√		√
Recovery of E Typhosis from:									
Blood	√			√	√				
Urine		√		√					
Stool	√	√	√				√	√	√

4,200,000 RBC., 8,100 WBC., 78 per cent neutrophiles, 22 per cent lymphocytes, 10 mm. sedimentation rate in one hour. Widal agglutination was positive in "H" and "O" antigens (1:320). Admission urine and feces cultures were positive for E. Typhosis. Subsequent laboratory procedures revealed urine cultures positive for E. Typhosis on the 7th and 19th hospital days. Widal was positive in "H" and "O" antigens (1:320) on the 4th and 8th hospital days. Feces cultures were positive on the 4th, 18th, 20th, 34th, 36th, and 40th hospital days. On admission the patient was started on sulfapyridine, grains xv, every four hours for a total

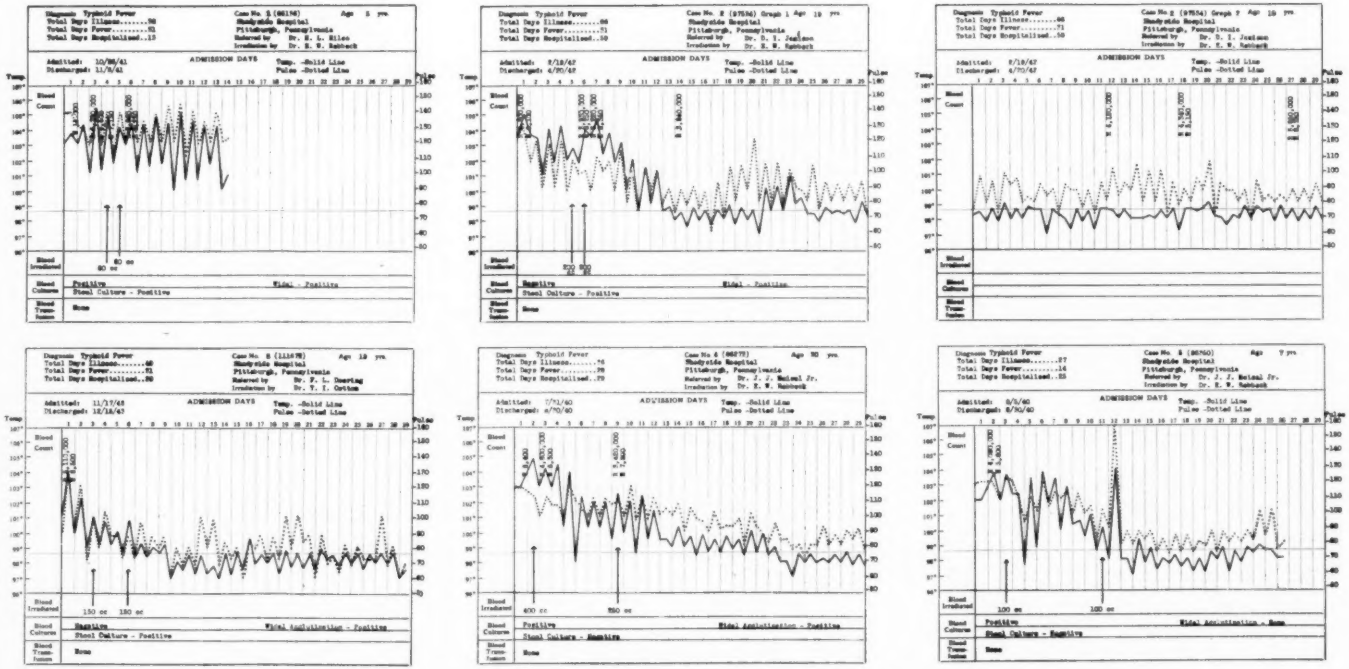

Fig. 1—Upper Left, Case 1. Upper Center and Right, Case 2. Lower Left, Case 3. Lower Center, Case 4. Lower Right, Case 5.

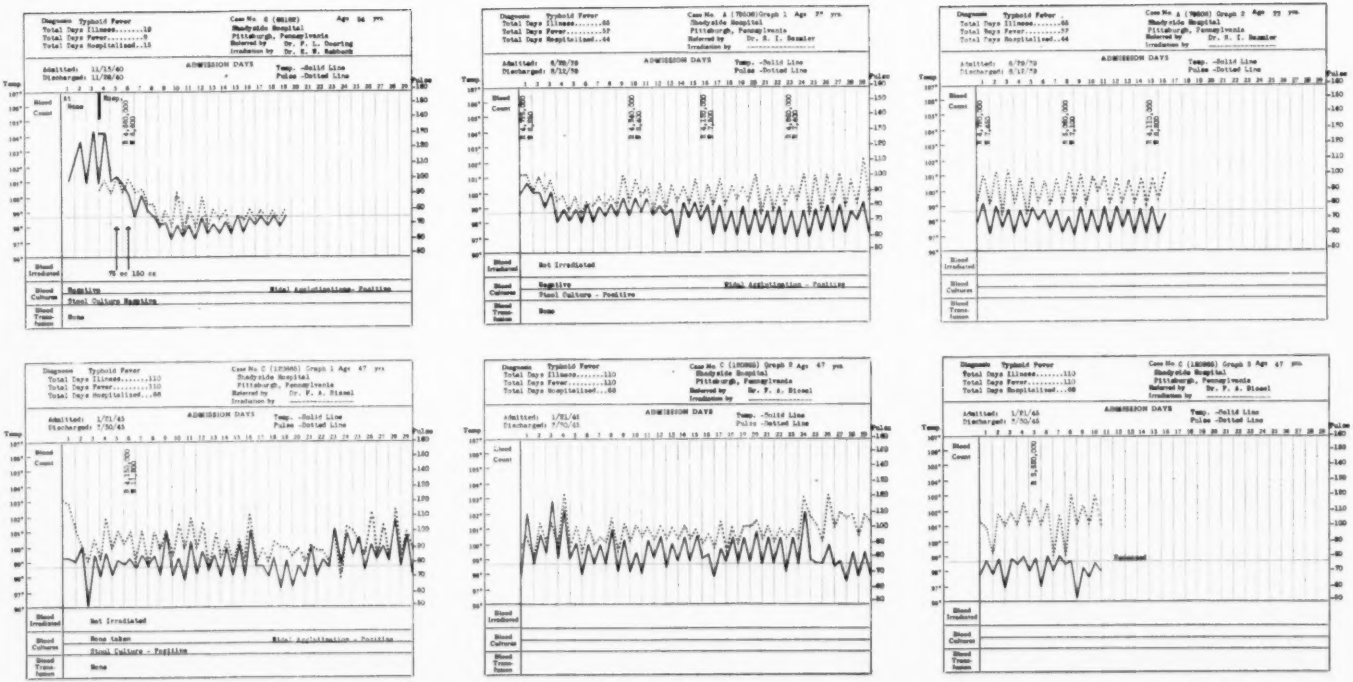

Fig. 2—Upper Left, Case 6. Upper Center and Right, Non-irradiated Case A. Lower Left, Center and Right, Non-irradiated Case C.

of 28 grams until diagnosis was established. Patient was then given UBI therapy on the 4th and 5th hospital days. Her temperature and pulse were normal on the 12th hospital day (19th day of illness) and remained so except for one relapse on the 20th hospital day, at which time the temperature was elevated to 101 for three days. There was prompt subsidence of all symptoms of relapse on the 22nd hospital day. Sulfaquanidine, grams 11, five times daily, was begun on the 34th hospital day because stool cultures were still positive. The sulfaquanidine was cancelled on 4/11/42, the 51st hospital day, following negative stools on the 46th, 47th, 49th and 50th hospital days. The patient was discharged as clinically recovered on 4/20/42, the 59th hospital day (66th day of illness).

Case 3:—Miss R., age 19, was admitted 11/17/43, with a three week history of chills, fever up to 106.3, diarrhea, nausea, and generalized abdominal pain. The admission temperature was 101.2, pulse 92 and respirations 20. Blood work on admission revealed 5,110,000 RBC., 6,500 WBC., 62 per cent neutrophiles, 33 per cent lymphocytes, 5 per cent monocytes, 1 per cent eosinophiles, 7 mm. sedimentation rate. Widal agglutination was positive in "H" and "O" antigens (1:320). Stool examination revealed the presence of E. Typhosis. The blood and urine cultures were negative. Sulfathiazole, grains xv, every four hours, was begun on the second hospital day for a total of 460 grains. UBI therapy was given on the third and sixth hospital days. The temperature was normal on the 10th hospital day (31st day of illness). Sulfaquanidine was begun on the 9th hospital day and continued for five days for a total of 450 grains. She was discharged as clinically recovered following five negative stools on the 28th hospital day (49 days from onset).

These three cases received a combination of sulfonamide therapy and UBI therapy and were discharged as clinically recovered on an average of 51 days from the onset of symptoms. The average time until the afebrile stage was reached from the onset of symptoms was thirty-one days. There was one relapse (in Case 2), which lasted only three days.

Case 4:—Mrs. L., age 30, was admitted on 7/31/40, with a one week history of severe headache, generalized abdominal pain and backache, vomiting and frequency of urination. Admission temperature was 103, pulse 120 and respirations 24. Admission blood work revealed 8,400 WBC., 76 per cent neutrophiles, 24 per cent lymphocytes, 48 mm. per hour sedimentation rate. Blood cultures were positive for E. Typhosis on 8/1 and 8/2/40. Widal agglutination on 7/31/40, revealed "H" antigen positive (1:40) and the "O" antigen (1:80). On 8/5/40, both "H" and "O" antigens were positive (1:320). Urinalysis on 7/31/40, revealed a trace of sugar and albumin, positive reaction to acetone and diacetic acid and positive culture for E. Coli and Staphylococcus albus. Patient was given ultraviolet blood irradiation therapy on the first and eighth hospital days. The temperature was normal on 8/22/40, the twentieth hospital day (28th day of illness), and she was discharged as clinically recovered the twenty-ninth hospital day (36th day of illness) with two successive negative stool cultures.

Case 5:—Miss L., age 7, (daughter of Case 4) was admitted on 8/5/40, with a two day history of headache, generalized pain in the abdomen and slight epistaxis. Admission temperature was 102, pulse 120 and respirations 24. Admission blood work revealed 4,090,000 RBC., 5,600 WBC., 78 per cent neutrophiles, 19 per cent lymphocytes, 3 per cent monocytes. Blood culture was positive for E. Typhosis. Urinalysis revealed a faint trace of albumin and positive reactions to acetone and diacetic acid. Patient was given UBI therapy on the 3rd and 11th hospital days. Temperature and pulse were normal on the 12th hospital day (14th day of illness), and the patient was discharged clinically recovered on the 25th hospital day (27th day of illness) with two successive negative stool cultures.

Case 6:—Mrs. B., age 34, was admitted on 11/13/40, with a four day history of frequency and burning on urination, general malaise, severe frontal headache, generalized abdominal pain and stiffness of neck, constipation and flatulence. Temperature had ranged between 101 and 102 since the onset 11/10/40. The Widal agglutination taken the day before admission showed "O" and "H" antigens

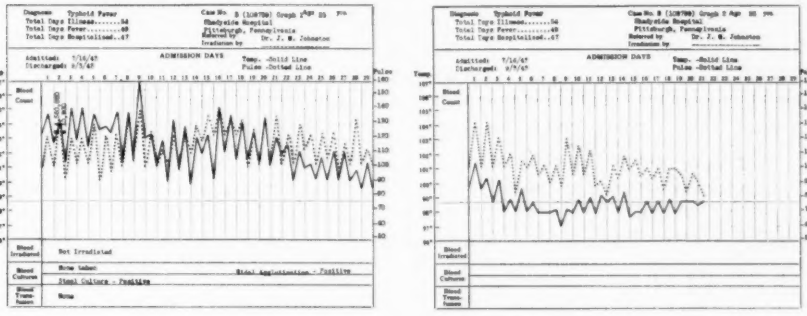

Fig. 3—Non-irradiated Case B.

positive (1:320). She had eaten contaminated homemade ice cream from which source three other cases developed in her immediate neighborhood. The admission temperature was 104.2, pulse 96 and respirations 24. The blood work revealed 4,660,000 RBC., 6,600 WBC., 73 per cent neutrophiles, 22 per cent lymphocytes, 5 per cent monocytes. The admission Widal agglutination was positive in "O" and "H" antigens (1:320). Urine, stools and blood culture were negative. The spleen was enlarged. She received UBI therapy on the 1st and 2nd hospital days. Two days later her temperature reached normal and stayed so. An interesting reaction occurred: on 11/13/40, Widal was positive in "O" and "H" antigens (1:320) as stated. On 11/16/40, twenty-four hours following the second irradiation, the Widal was reported negative in "O" and "H" antigens (1:320). On 11/18/40, Widal was reported positive in "H" antigen (1:320) and negative in "O" antigen (1:320). As stated, her temperature reached normal on 11/17/40, the 4th hospital day (8th day of illness). After two successive negative stool cultures, she was discharged on 11/28/40, the 15th hospital day (19th day of illness) and made an uneventful recovery. Stool examinations at home remained negative.

These three cases had no other specific therapy than UBI and were discharged as clinically recovered on an average of twenty-four days from the onset of the symptoms. The average time until the afebrile stage was reached from the onset was sixteen days.

The six cases in the first two groups averaged twenty-three days of illness and were discharged as clinically recovered in an average of thirty-nine days.

Cases Not Irradiated

Case A:—Mr. M., age 33, was admitted on 6/29/39, with a three week history of general malaise, sweating, lassitude, chills, fever, continuous muscular aching and headache, loss of appetite and weight and cough. There was no diarrhea. The patient had been in the Bahamas when illness began. Admission physical examination was not remarkable. Admission temperature was 100, pulse 104, respirations 20. Admission blood work revealed 4,725,000 RBC., 6,250 WBC., 60 per cent neutrophiles, 36 per cent lymphocytes, 4 per cent monocytes, 45 mm. sedimentation rate in one hour. Widal agglutination was positive in "O" antigen (1:320) and "H" antigen (1:640). Urine and stool cultures were negative. The patient ran a low-grade temperature until 7/30/39, the 31st hospital day (52nd day of illness). One stool culture was positive for E. Typhosis 7/25/39. His recovery was uneventful, and he was discharged on 8/12/39, the 44th hospital day (in the 10th week of illness). He received no specific therapy.

Case B:—Miss G., age 25, was admitted 7/16/43, with a one week history of fever, weakness, diarrhea and general malaise. Physical examination revealed tenderness in the right upper abdominal quadrant. Admission temperature was 103.2, pulse 100 and respirations 28. Admission blood work revealed 4,460,000 RBC., 10,900 WBC., 75 per cent neutrophiles, 23 per cent lymphocytes, 2 per cent monocytes. Widal agglutination was positive in "O" antigen (1:320) and "H" antigen (1:640). Repeat Widal agglutination on 7/19/43, the third hospital day, revealed the same results as above. Stool culture on 7/19/43, the third hospital day, revealed E. Typhosis. The patient ran a typical typhoid course of temperature, toxemia, abdominal symptoms, muscular twitchings, convulsive tremors and restlessness. She became afebrile on 8/28/43, the 42nd hospital day (49th day of illness). She was discharged on 9/3/43, the 47th hospital day (54th day of illness), following repeated negative stools. She received no specific therapy.

Case C:—Mrs. C., age 47, was admitted 1/21/45, with a six week history of weekness, loss of weight, constipation and nausea but no vomiting. Stools had been clay colored and associated with much mucus. On admission physically she was jaundiced, lethargic and depressed with tenderness and rigidity in the upper right quadrant. Admission temperature was 99.2, pulse 120 and respirations 24. Admission blood work revealed 4,150,000 RBC., 11,200 WBC., 66 per cent neutrophiles, 24 per cent lymphocytes, 7 per cent monocytes, 3 per cent eosinophiles. On 2/20/45, the 31st hospital day, Widal agglutination revealed "O" and "H" antigens positive (1:640). Stool culture also revealed E. Typhosis on two specimens; and again, low-grade fever with increasing toxicity. The liver became en-

larged, and jaundice visible. Abdominal cramps and diarrhea manifested early in hospitalization and persisted intermittently. Sulfaquanidine, grams v, T.I.D., was begun 2/7/45, the 17th hospital day, for three days; then grams II, every six hours through 2/17/45, for a total of 70 grams. Penicillin was started on 3/16/45, 20,000 units every three hours for a total of 1,000,000 units. Patient apparently succumbed to an overwhelming toxemia, expiring on the 68th hospital day in the 16th week of her illness.

The three control cases reveal an average of 70 days of fever and 78 days to discharge (which includes one death who lived for 110 days from the onset of symptoms).

TABLE II. SUMMARY

	Case	Total Days Illness	Total Hosp. Days	Total Days Fever	Total Days Fever After UBI	Outcome
	Irradiated Cases					
Irradiation and Sulfa Therapy	#1	38	13	31	24	Recovery
	#2	66	59	31	19	Recovery
	#3	49	28	31	6	Recovery
Irradiation Therapy Only	#4	36	29	28	20	Recovery
	#5	27	25	14	10	Recovery
	#6	19	15	8	2	Recovery
	Nonirradiated Cases					
	#A	65	44	52		Recovery
	#B	54	47	49		Recovery
	#C	110	68	110		Death

Rationale

The successful outcome of these cases may be explained by the following known actions of ultraviolet blood irradiation therapy:

1. Increase in the oxygen combining power of venous blood[1].
2. Increase in the opsonic index[2].
3. The consistently observed peripheral vasodilating effect[3, 4].
4. The ability of ultraviolet light to inactivate toxins and viruses[5, 6].

Summary

1. Nine cases of Typhoid Fever diagnosed by: (1) histories, (2) blood counts, (3) agglutination tests and/or the recovery of the Typhoid Bacillus in either blood, stool or urine were reported in this series (See Table II).

2. Three cases were treated by the combination of Blood Irradiation Therapy (Knott Technic) and sulfonamides. Three cases were treated by Blood Irradiation Therapy alone. The remaining three cases were treated symptomatically (one had sulfonamide and penicillin therapy).

3. The three cases treated by the combination of sulfonamides and UBI reached the afebrile stage in an average of thirty-one days and were discharged as clinically recovered in an average of fifty-one days.

4. The three cases treated by UBI alone responded in an average of sixteen days until the afebrile stage was reached and an average of twenty-four days until clinically recovered.

5. Combining these six cases, there is an average of twenty-one days until the afebrile stage was reached and an average of thirty-nine days until clinically recovered. There were no deaths in this series. The detoxifying effect of this therapy frequently seen in the past was consistently observed during the course of these cases.

6. The three cases not treated by UBI had a normal temperature in an average of seventy days from the onset of symptoms and were discharged in an average of seventy-eight days (with one death, that patient living for 110 days).

Conclusion

Ultraviolet blood irradiation therapy (Knott technic), particularly when used alone, is of distinct value in the treatment of typhoid fever.

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Discussion

Dr. Gerald C. Grout (Dayton, Ohio):—The presentation of these nine cases of Typhoid Fever, six successfully treated with the Knott technic of Ultraviolet Blood Irradiation, is of great interest to me as I have one case to present which was treated by the same method with favorable results.

Case Report

The patient, a white female student nurse, age twenty-three years, was first seen by me six weeks following the onset of her illness.

At that time the blood culture, stool culture and urine culture were positive for Eberthela Typhosa.

Blood agglutination studies with Typhoid "H" were positive in dilutions up to 1:6400 and studies with Typhoid "O" were positive in dilutions up to 1:400.

The patient was having severe chills every eight hours lasting for about one hour, followed by temperature elevations as high as 105.4.

Medication over the six weeks' period had included large doses of Penicillin, large doses of Streptomycin, frequently repeated small blood transfusions and other general supportive measures. The patient had shown no improvement as a result of any of this treatment.

At the end of six weeks the patient had lost twenty-five pounds in weight and her general condition was moribund.

On May 14, 1948, which was the beginning of the seventh week of illness, the patient was given an Ultraviolet Blood Irradiation treatment by the Knott technic. This treatment was repeated the next day on May 15, 1948, and again on May 19, 1948. At the end of forty-eight hours following the first Blood Irradiation treatment, or, in other words, twenty-four hours after the second Blood Irradiation the chills stopped. At the end of ninety-six hours following the first Blood Irradiation the patient's temperature fell to normal and remained there. Furthermore, the patient's blood culture immediately became negative, her general condition improved and at the end of ten days was out of bed.

Even though the patient made a very rapid recovery following Blood Irradiation treatment, the stool culture did not become negative until four weeks after the first Blood Irradiation was given.

A SIMPLE BENZIDINE TEST FOR OCCULT BLOOD IN FECES

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It is a common experience of laboratory workers using the ordinary laboratory procedures[1] to encounter many factors other than occult blood producing positive reactions of stools in patients with suspected gastrointestinal bleeding. Ogilvie[2] believed that iron compounds might not interfere with benzidine tests. This belief was apparently confirmed by Schwartz and Vil[3]. However, Johnson and Oliver[4] found false-positive benzidine reactions following administration of considerable amounts of meats. As a result of this state of confusion and the increased number of false-positive reactions observed by the authors through years of experience in clinical laboratories and in private practice (MBL), they devised a technic eliminating known false-positive reactions, for more accurate results.

It is the purpose of this paper to evaluate the results of the ordinary laboratory procedure in comparison with ours.

Materials and Methods

All fecal samples used in this study were collected routinely in clean containers. A small portion of the feces is emulsified in distilled water, then passed through filter paper (Whatman #5 or more retentive paper) into a clean test tube to obtain a clear filtrate. If clouded due to breach in the filter paper, refilter. To 3 cc. of the filtrate are added 8 drops of 50 per cent aqueous acetic acid or glacial acetic acid, with mixing. To the resulting mixture are added 8 drops of hydrogen peroxide (C.P., 3 per cent), followed by shaking. Overlay the final mixture with an alcoholic benzidine solution*, drop by drop, to obtain a contact ring. This is tilted to bring about slight mixing at the ring. A positive reaction is indicated by the appearance of a greenish ring at the area of contact of the benzidine solution and the filtrate mixture, spreading into the benzidine solution either immediately or up to 2 minutes time, the intensity or gradient of color being in relation to the concentration of blood.

When testing for blood in vomitus and gastric contents, water is added to either in half the volume of material used, the mixture is filtered, and the test is carried through as previously. In urine, when only a few RBC are found microscopically (4 to 8 RBC per HPF), the specimen is centrifugalized or sedimented, the bottom 0.1 cc. containing the sediment is transferred to a clean test tube, 0.4 cc. of distilled water is added, and the test is carried through as previously.

*Dissolve 6 grams of benzidine base C.P. powder (for occult blood) in 100 cc. of ethyl alcohol, 95 per cent, and preferably allow to age 2 or 3 days before using.

Results and Analysis

In the course of our studies, in a variety of conditions of ulcerative and non-ulcerative gastrointestinal types, approximately 32 per cent of the specimens gave positive benzidine tests with the authors' method, while 68 per cent gave positive reactions with the ordinary method. *Gastrointestinal carcinoma specimens gave 99+ per cent positive reactions with both methods.*

The maximum sensitiveness of our method was found to be 1:100,000 with hemoglobin, C.P.

Summary and Conclusion

Our method results in the elimination of most of the substances producing false-positive reactions as follows: (a) filtration of the stool suspension eliminates blood-containing particles of meats; oxidase-containing particles of foods; particles of animal charcoal; and insoluble metal salts (of iron, copper, etc.) and (b) the acetic acid destroys soluble oxidases.

Furthermore, in our method the contact layer or ring test results in the soluble hemoglobin compounds giving true positive blood reactions in dilutions up to 1:100,000.

Meat-free days are unnecessary preceding our test for occult blood.

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CHAPTER ACTIVITIES

Argentina Chapter

A meeting of the Argentina Chapter of the National Gastroenterological Association was held in Buenos Aires on 24 June 1949.

Dr. Carlos Reussi, distinguished Gastroenterologist of the Hospital Rivadavia and author of several important scientific publications was elected President to succeed Dr. Arturo Richieri.

NEWS NOTES

Executive Board Meeting

A meeting of the Executive Board of the National Gastroenterological Association was held at the headquarters office, New York City on 27 June 1949.

Routine administrative correspondence was presented and ordered properly disposed of.

The following new members were elected to the National Gastroenterological Association: Dr. Carl DePrizio, Attleboro, Mass., Member; Dr. Lawrence Francis Cozza, Medford, Mass., Member; Dr. Aurel Gilbert Lavoie, Springfield, Mass., Associate Fellow; Dr. Adolph Abraham, New York, N. Y., Fellow; Dr. Max S. Konigsberg, New York, N. Y., Member; Dr. Philip Laden, New York, N. Y., Associate Fellow; Dr. Max Markowitz, Forest Hills, N. Y., Member; Dr. Saul A. Schwartz, Bronx, N. Y., Associate Fellow; Dr. Esther Tuttle, New York, N. Y., Member; Dr. Victor Willner, New York, N. Y., Associate Fellow; Dr. Irving A. Beck, Providence, R. I., Fellow; Dr. Boston M. Day, San Francisco, Calif., Associate Fellow; Dr. Max Harold Ruby, Waterbury, Conn., Fellow; Dr. Harold Bartlett Johnson, Buffalo, N. Y., Member and Dr. Bahaeddin Safavi, Mt. Vernon, N. Y., Member.

Dr. Morrison reported that all the speakers for the Convention had accepted and there would be 18 papers. Letters were being sent to members of the Association inviting them to discuss the papers being presented.

Dr. Morrison also reported that the course in Gastrointestinal Surgery will consist of 31 papers to be presented, and that one-half the quota of 250 applications had already been received.

Dr. Johnsen reported for the Committee on Standards and Rating that a follow-up to the questionnaire had been sent out to the hospitals which have not responded and that the returns were gratifying.

The matter of insignia for Fellowship was again taken up and the sketch of a key was approved and ordered to be ready for distribution, at the Convention.

The Prize Award Contest Judges submitted their report on the contest papers and the announcement of the winner will be made in the very near future.

In Memoriam

We record with profound sorrow the passing of Drs. Arthur B. Donovan, Member, Boston, Mass.; Jorge R. Muniz, Fellow, Havana, Cuba and Ernest E. Smith, Associate Fellow, New York, N. Y.

We extend our deepest sympathies to the families of the deceased.

ABSTRACTS

Gastrointestinal Tract

SPINDLE-CELL TUMORS OF THE GASTROINTESTINAL TRACT. William L. Palazzo and Milford D. Schulz. Radiology. **51**:779, (Dec.), 1948.

Spindle-cell tumors of the gastrointestinal tract were found in 0.67 per cent of 12,000 necropsies and in 1.2 per cent of 5,313 surgical records at the Massachusetts General Hospital. Both benign and malignant varieties occurred with increasing frequency as age progressed. Of the total 140 spindle-cell tumors, 9 per cent were located in the esophagus, 65 per cent in the stomach, 17 per cent in the small intestine, and 9 per cent in the colon. Spindle-cell tumors accounted for 2.3 per cent of all tumors surgically removed from the esophagus in the past 16 years, for 5 per cent of those in the stomach, for 7 per cent of those in the small intestine, and for 0.2 per cent of those in the colon. Of those spindle-cell tumors found in the esophagus, 33 per cent were malignant, in the stomach, 36 per cent were malignant, and 54 per cent of the patients bearing the tumors remained well and free of disease for an average of 8 years. Recurrence or metastasis, if present, was evident within 4 years. Metastasis to the liver and lymph nodes occurred in 33 per cent of the malignant gastric tumors. In the small bowel 77 per cent were malignant; none of the patients remained free of disease for longer than 3 years. Eighty-six per cent of the colon tumors were malignant, only one patient of this group is alive and there has been no sign of recurrence for 5 years. There are no safe criteria by which the benignity of such a tumor may be recognized with certainty.

Franz J. Lust

Stomach

PRESENT STATUS OF THE TREATMENT OF PEPTIC ULCER. Waltman Walters, B. C. Brownson and S. K. Phillips. J. Michigan M. Soc. **48**:202-209, (Feb.), 1949.

The authors refer to their present series of 177 cases of vagotomy up to December 31, 1947. They believe that the best approach to the vagus at the lower end of the esophagus was in most cases through an abdominal incision. Exploration of the abdomen is possible, and in five cases, the presence of a peptic ulcer was not substantiated at operation, and so vagotomy was not performed. In ten cases, associated abdominal lesions, such as cholelithiasis, Meckel's diverticulum or inflammation of the appendix, required removal in addition to the vagotomy for peptic ulcer. In five of 77 cases in which Walters performed vagotomy, recurrent ulceration has developed. In four of these five cases the results of insulin tests were negative. The authors evaluate the results of vagotomy without other surgical procedures on the stomach and duodenum, gastrojejunal ulcer after gastroenterostomy, and after partial gastrectomy, duodenal ulcer after vagotomy and gastroenterostomy, vagotomy, pyloroplasty and excision of ulcer, and gastric ulcer (partial gastrectomy).

The authors believe vagotomy is clearly indicated in cases of recurring ulceration after partial gastrectomy.

Hyman I. Goldstein

MASSIVE HEMORRHAGE FROM CARCINOID OF THE STOMACH. V. E. Scherman, M. Hara and H. F. Trafton. J. Missouri M. A. **46**:175-177, (Mar.), 1949.

"Carcinoid Tumors", is a term first applied by Oberndorfer in 1907, to tumors arising from the chromo-argentaffin cells of Masson in the intestinal tract. Gosset and Masson have shown that certain granules in the cytoplasm in the Masson cells reduced silver and appeared as tiny brown or black particles. These tumors are sometimes called "argentaffin tumors". Silver reducing granules, are not always present in carcinoids.

These authors report two cases, a 48 year old housewife, and a man, aged 65 years. The second case of carcinoid of the stomach was found incidentally at autopsy.

Hyman I. Goldstein

AN ANALYSIS OF X-RAY FINDINGS IN 405 CASES OF BENIGN GASTRIC AND PYLORIC ULCERS. Walter A. Russell, Sydney Weintraub and Harold L. Temple. Radiology. **51**:790, (Dec.), 1948.

The confusion in anatomical terminology in reference to the stomach is emphasized, and a plea is made for the adoption of precise nomenclature to be used and understood equally well by the roentgenologist, surgeon and pathologist.

Of 429 ulcers in 405 cases, 65 per cent occurred on the lesser curvature of the body of the stomach, including the region of the incisura angularis. A relatively high number, 8 per cent, were located in the prepyloric area. 19 occurred at the pylorus. The radiographic criteria for diagnosis of pyloric ulcers are given. The reason for the discrepancies between the radiographic and surgical location of a lesion, particularly in the region of the pylorus, are discussed. It is the opinion of

the authors that the x-ray diagnosis of location is by far the more accurate. A comparison is made between the results of the medical and surgical treatment in patients requiring hospitalization. The treatment of choice is gastric resection, which showed 92 per cent good results as compared with 41 per cent for medical management. Multiple gastric ulcers were observed in 23 cases (5.7 per cent). Triple ulcers were seen once. In the case of triple ulcers, and in 10 instances of double ulcers, the lesions were observed simultaneously. Duodenal ulcers, either active or healed, were associated with gastric ulcers in 10 per cent. FRANZ J. LUST

VAGOTOMY FOR PEPTIC ULCER. J. E. Summers. J. Michigan M. Soc. **48**:209-212, (Feb.), 1949.

Vagus nerve section, usually partial, has been performed sporadically for many years for a variety of abdominal conditions.

Subtotal gastrectomy is generally approved as the best method of treatment for chronic gastric ulcer. Partial gastrectomy for duodenal ulcer is not an entirely satisfactory procedure because of the magnitude of the operation, the mortality rate, the development of jejunal or stomal ulcers, nutritional disturbances, and the frequent occurrence of the "dumping syndrome".

The gastric glands, containing the chief and partial cells, occur over the entire surface of the stomach, except the cardia and the pylorus. The parietal cells secrete hydrochloric acid while the chief cells secrete pepsin. The author discusses physiology of gastric secretion, cause of peptic ulcer, operative technic, and vagus nerve section for peptic ulcer. Vagus nerve resection, by interrupting the cephalic (psychic) phase of gastric secretion, diminishes the acidity of the gastric secretions, and rests upon a sound physiological basis. The clinical results are unusually favorable.

HYMAN I. GOLDSTEIN

CHRONIC ULCERATIVE COLITIS. Walter Lincoln Palmer. Gastroenterology. **10**:767-781, (May), 1948.

Dr. Palmer has had extensive experience with chronic ulcerative colitis, and published several interestingly informative articles on the subject in recent years.

Inflammatory and ulcerative diseases of the colon has been a difficult problem for centuries. Many of the older writers had to contend with many such cases encountered in their practice.

The severity of the symptoms and the extent of the disease process do not always show a direct relationship. Profuse bleeding and tenesmus may occur with a localized lesion. Extensive disease may be discovered in an apparently well person passing normal or several soft stools daily.

Palmer refers to Bargen's streptococcic theory, to Felsen's work, and to the reports by Brown (1946), Brown and Bargen (1938), Kiefer (1932), Dock, Dragstedt and others—and suggests that "more work on the bacteriologic (etiologic) phase of the disease is clearly needed". Palmer then discusses the psychogenic theory—how much of a role do the emotions play and under what circumstances.

HYMAN I. GOLDSTEIN

X-RAY OBSERVATIONS BEFORE AND AFTER VAGOTOMY. Warren W. Furey. Radiology. **51**:806, (Dec.), 1948.

The radiologist should be familiar with the vagotomy controversy and with the pertinent clinical and laboratory findings and thus know what to expect when asked to study one of these patients. Furey demonstrates some of the occasionally startling and paradoxical roentgen findings. It is rather striking to have a patient appear well, state that he feels fine, has no distress, eats everything, sleeps well and has gained weight since operation, and then to find, that his stomach is markedly distended, contains considerable quantities of retained food material and retains opaque material almost completely, in some instances over a 24 hour period; to have great difficulty in visualizing the duodenal bulb and, when it is demonstrated, to observe a marked deformity; sometimes to find evidence of a persisting ulcer crater in the complete absence of pain or tenderness on direct palpation. FRANZ J. LUST

TUBERCULOSIS OF THE STOMACH AND DUODENUM. Herman W. Ostrum and W. Serber. Am. J. Roentgenol. **60**:315-322, (Sept.), 1948.

Tuberculous lesions of the stomach and duodenum are uncommon.

Tuberculous mesenteric lymph nodes may be the primary lesion. The routes of infection may be through the blood stream; by lymphatic channels; directly through the mucosa, and spread by continuity from adjacent lymph nodes or other structures. Why the rarity of gastric and duodenal tuberculosis when tuberculosis of the intestines and rectum, and peritoneum is not uncommon?

The authors suggest that gastric acidity, intact mucosa, scarcity of lymphoid structures in the gastric wall, rapid emptying time of the stomach and inherent resistance, may explain this rarity of gastric and duodenal tuberculosis.

Single or multiple ulcers (often deforming in character) are found in the duodenum. The second and third portions are more frequently involved—perforation and fistulae may occur. Frequently, tuberculosis occurs elsewhere in the body. In almost all recorded cases enlarged mesenteric and retroperitoneal tuberculosis lymph nodes are present. The x-ray appearance is that of cancer of benign ulcer.

The authors report three cases, a white women, 45 years old; a negro, 25 years old, and a negro, 43 years old. All three were carefully examined at necropsy—by Doctors E. Mertens, W. E. Ehrich, Valdes-Dapena, and Dale R. Coman, of the University of Pennsylvania. Two cases showed evidence of tuberculosis of the stomach, and one case showed duodenal involvement. None of these cases had evidence of active pulmonary tuberculosis. The absence of pulmonary tuberculosis should not exclude the possibility of gastroduodenal tuberculosis. HYMAN I. GOLDSTEIN

INTESTINES

PRIMARY RESECTION OF THE COLON IN ULCERATIVE COLITIS. G. Gavin Miller, Campbell McG. Gardner and Charles B. Ripstein. Canad. M. A. J. **60**:584-585, (June), 1949.

The authors report 24 cases treated surgically. Ileostomy alone is seldom adequate. In most cases, permanent ileostomy is necessary. The authors advise ileostomy combined with right hemicolectomy as an initial operation, and this is followed within three to six months by resection of the remaining colon. The authors report 24 consecutive cases of this procedure without a mortality. The rationale in this treatment is that the right colon is the absorptive part of the large bowel, and its removal at once eliminates the absorption and bacterial toxins. The right bowel also represents the area from which the loss of protein and blood is noted.

The technic of the operation as described by the authors consists of: The entire colon to the level of the lower sigmoid is resected at one stage. A terminal ileostomy is made six cm. below and three cm. to the right of the umbilicus. The distal sigmoid segment is exteriorized through a stab wound in the left lower quadrant. No attempt is made to invert or anastomose bowel ends. The authors feel that primary section of the colon is the operation of choice in ulcerative colitis. With proper pre- and postoperative care, it carries a lower mortality and morbidity than an ileostomy alone. It has the advantages of immediate removal of the diseased bowel and it eliminates multiple stage operations. LIONEL MARKS

THE DIAGNOSIS AND PATHOGENESIS OF TROPICAL SPRUE. Mario Stefanini. Acta Med. Scandinav. **133**:114, 1949.

This paper presents considerations on the diagnosis and pathogenesis of the sprue syndrome derived from a review of studies carried out in British India during the war, on almost 6,000 cases of tropical sprue occurring among military units stationed there.

The fundamental symptoms of the sprue syndrome is represented by a partial defect of intestinal absorption earlier and particularly well detectable in the case of fats. The progression of the untreated clinical picture is closely related to the intestinal malabsorption and can be divided in three successive stages. A first in which the initial signs of impaired intestinal absorption predominate (diarrhea with steatorrhea, asthenia, dyspepsia, meteorism); a second in which secondary deficiency signs (glossitis, stomatitis, etc.) set in and a third in which, with the appearance of macrocytic anemia, the patient presents a full blown picture of tropical sprue. The first two stages include also the mild and incomplete forms of the sprue syndrome variably designated at the present time (hill diarrhea, parasprue, nutritional diarrhea etc.).

In the authors experience the disease was found as frequently in Indians as in Europeans. A partial defect of intestinal absorption of fats, usually detected earlier and more easily than that of any other dietary constituents, represents the basis for the diagnosis of the sprue syndrome. The "fat balance technic" described in the paper seems to represent the best method of study of intestinal absorption. Chemical analysis of the feces usually shows high steatorrhea with split/unsplit fat ratio higher than normal. The greatest part of split fecal fat in sprue appears to be present as soaps.

Dietary deficiencies are the most important factors among those leading to the development of tropical sprue. Alone, however, they do not explain all the peculiarities of the series of cases described in India during the war such as the epidemic occurrence of the disease in a particular period of the year and the sudden "explosive" high incidence among the military shortly after their transfer to special areas. It appears that sprue occurs almost epidemically whenever and wherever, besides the dietary inadequacy, favorable seasonal, local and climatic conditions exist. The nature and mechansim of action of these pathogenetic factors are at present unknown.

FRANZ J. LUST

MALIGNANT TUMORS OF THE COLON AND RECTUM. R. W. Postlethwait. Ann. Surg. **129**:34-46, (Jan.), 1949.

Malignant tumors of the colon and rectum were found at Duke Hospital in 441 patients during the 15 year period 1931-1945. The incidence, symptomatology, treatment and results in this group are analyzed and reported by the author.

Twenty and one-half per cent of this group gave a family history of malignant tumor. A number (31) of these patients had operations before coming to Duke Hospital. Four had (previously) biopsy of rectal lesions.

The infrequency of biopsy is to be noted. Hemorrhoidectomy or repeated injections for hemorrhoids were carried out in 9 per cent of patients with lesions of the rectum and rectosigmoid.

The duration of symptoms, from the onset of the first symptom to time of reporting to the hospital, averaged 8.7 months for the entire group. Abdominal pain was the most frequent symptom in both left and right colon groups and not so common in the rectum and rectosigmoid group. In this latter group of 220 patients melena, constipation, tenesmus and diarrhea were the most frequent symptoms in this order. Abdominal pain, mucus in stool, and decreased calibre of stool, follow in this order. Of 229 cases of rectal malignancy, the lesion was felt by digital examination in 222, not felt but seen by proctoscopy in 5, and no examination recorded in 2.

The radiologic findings were positive by barium enema in 81 per cent of right colon lesions and 88 per cent of left colon lesions.

Early diagnosis of malignant lesions of the colon and rectum is one of the most important factors in increasing resectability.

Appropriate measures must be utilized preoperatively to correct fluid and electrolyte balance, hypoproteinemia, vitamin deficiency, and anemia. Improved surgical technic blood transfusion at operation, better anesthesia, the antibiotic and chemotherapeutic agents and gastrointestinal intubation have decreased morbidity and mortality.

The procedures of choice at Duke Hospital at present are resection and primary anastomosis for lesions of the colon, and combined abdominoperineal resection for tumors of the rectum.

HYMAN I. GOLDSTEIN

PATHOLOGY AND LABORATORY RESEARCH

THE MECHANISM OF THE HYPOGLYCEMIC ACTION OF ALLOXAN. Mervin Griffith. Australian J. Exper. Biol. & M. Sc. **26**:339, (July), 1948.

Guinea-pig pancreas is shown to have as large a store of insulin as that of the rabbit. Alloxan has no hypoglycemic action when injected subcutaneously or into the bloodstream of normal guinea-pigs. Hypoglycemia does occur, however, when alloxan is injected directly into the pancreas of this species. The absence of hypoglycemia cannot be attributed to an insufficient store of preformed insulin. Evidence is given which supports the possibility that the absence of hypoglycemia is due to the relatively high blood reduced gluthathione in guinea pigs. Alloxan hypoglycemia does not occur in recently pancreatectomized rabbits. The conclusion from the above data is that hypoglycemia due to alloxan is pancreatic in origin.

FRANZ J. LUST

DETOXIFICATION OF BENZOIC ACID BY GLYCURONIC ACID UNDER NORMAL CONDITIONS AND IN LIVER DISEASE. Bengt Borgstroem. Acta Med. Scandinav. **133**:7, 1949.

The author presents his findings in an investigation into the detoxification of benzoic acid, with special reference to the detoxification by glycuronic acid, partly in human subjects and partly in rats. The investigation gave the following results: 1) The excretion of benzoyl-glycuronic acid in the urine seems proportional to the benzoic acid concentration in the organism, both in man and in rat. Free benzoic acid is excreted in the urine of rats, if more than 350 mg. benzoic acid per kg. rat is administered. 2) In cases of liver disease in man the benzoyl-glycuronic acid synthesis seems to be disturbed later than the hippuric acid synthesis, but the increase in the benzoyl-glycuronic acid excretion observed in connection with light liver damage is too small to warrant any further diagnostic conclusions on the basis of administration of benzoic acid according to Quick.

FRANZ J. LUST

LIVER AND BILIARY TRACT

THE DISTRIBUTION OF RED BLOOD CELL DIAMETERS IN LIVER DISEASES. Gerhard Larsen. Acta Med. Scandinav. **132**: Suppl. 220, 1948.

The clinical material used in the investigation of the blood cell diameter in liver disease is reported. It consists of 26 cases of acute hepatitis (duration less than 90 days with full recovery), of 32 cases of chronic hepatitis (duration of more than 90 days), and of 14 cases of cholecystitis or cholelithiasis (Obstructive jaundice). After a brief discussion of the age/sex incidence it is shown that all cases with hepatocellular damage have an increased diameter of the red blood cells, while the mean corpscular volume remains normal.

The cause of the macrocytosis is said by some authors to be changes in the peripheral blood stream. This hypothesis is discussed, and it is conclusively shown that neither jaundice, nor osmotic disturbances, nor any other peripheral cause previously suggested, can account for the findings in the present material. It is shown that the macrocytosis is probably due to the appearance

of a distinct new group of larger blood cells in the peripheral blood stream, so that the blood cell population becomes heterogeneous, consisting of a mixture of normal and pathological large cells.

Further arguments for the blood cell population being heterogeneous are given. Based on the present material it is calculated that the mean diameter of the hypothetical large cells must average 0.75 mg. higher values than those of the normal cells. Subsequent statistical analysis of the frequency curves, 412 in all, shows this assumption to be correct. Further proof is given that the analysis reveals real conditions in the pathological blood samples. It is shown that the pathological large cells are thinner than the normal and more resistant to hypotonic salt solutions.

Regeneration of normocytes in acute hepatitis occurs at the same rate as the formation of erythrocytes in pernicious anaemia, and that regeneration occurs according to the equation for autocatalytic mono-molecular reactions, the biological growth formula.

The frequency curves obtained in liver diseases are compared with the frequency curves from pernicious anaemia. It is shown that these curves are not identical: While the blood cell population in hepatitis consists of 2 components, the blood cell population in untreated pernicious anemia consists of at least 3, probably 4 components. When patients with pernicious anemia are treated with liver extract, the blood cell population changes so that it can be distinguished from that seen in hepatitis. This condition persists for months and years in cases of pernicious anemia, even when the blood counts become normal.

It is concluded that the disturbances seen in hepatitis as well as those seen in pernicious anemia are due to a disturbed maturation of the red blood cell precursors in the bone marrow. In pernicious anemia, the main disturbance lies at the level of the proerythroblasts, which partly develop into megaloblasts and megalocytes. This disturbance is removed when adequate treatment with liver extract is given.

In liver disease, as well as in treated pernicious anemia, the disturbance lies at the level of the macroblasts, which develop into macrocytes instead of normally, into normoblasts and normocytes. The cause of this disturbance in the maturation is discussed. Reports made by earlier authors may suggest that lack of a member of the Vitamin B group may be the cause. Some tentative experiments are reported which may indicate that niacin, in one way or another, plays a role. The results are however too conflicting to be decisive. FRANZ J. LUST

SPLEEN

PORTACAVAL SHUNTS IN THE TREATMENT OF PORTAL HYPERTENSION. AN ANALYSIS OF 15 CASES. R. R. Linton Hardy Jr. and Volwiler. Surg. Gynec. & Obst. **87**:129-144, (Aug.), 1948.

Linton et al. review 15 cases with special reference to the suture type of end-to-side splenorenal anastomosis with splenectomy and preservation of the kidney.

Portal hypertension develops secondary to partial or complete obstruction of the portal blood flow in human patients. The site of the block may be either in the liver, the intrahepatic type, or in the portal venous system, the extrahepatic type, as shown by Whipple. Esophageal varices develop as a result of either type of block.

Blood is shunted around the site of the obstruction through these channels (collateral) from portal to systemic venous system. Fatal hemorrhage may occur from ruptured esophageal varices.

Patients with portal hypertension secondary to a portal bed block, have sudden massive hemorrhage (hematemesis) and frequently, this is the first episode, that forces the patient to seek medical aid. Occasionally, melena occurs. Splenomegaly (congestive), and only occasionally hepatic enlargement, with secondary anemia, further point to this condition. Liver function tests in intrahepatic block, show high retention of bromsulfalein, positive cephalin flocculation test, elevated prothrombin time and reversal of albumin-globulin ratio. In extrahepatic block liver function tests are usually normal. HYMAN I. GOLDSTEIN

SPLENIC PANHEMATOPENIA. Prof. S. Van Creveld. Arch. Dis. Childhood. **23**:163-170, (Sept.), 1948.

The discovery of the Rhesus factor (Rh) has elucidated the nature of hemolytic disease of the newborn. In recent years, several other questions have arisen in regard to hemolytic anemias: such as the question of congenital and acquired forms of hemolytic jaundice (Loutit and Mollison, 1946—confirming Widal's idea of the familial form and the acquired form). Bergenheim and Fahraeus (1936) demonstrated lysolecithin as being formed and acting in the spleen, when the blood is slowed (or sludged?) in passage through this reservoir. The author refers to the role of the spleen in hemolytic anemias, and to the work of K. Singer ("Hypersplenism", J. Lab. Clin. Med., **30**:784, 1945) and Doan & Wright (Blood **1**:10, 1946). The author distinguishes between primary splenic panhematopenia and other anemias. This disease is characterized by a striking decrease in erythrocytes, leucocytes, and platelets, and can be differentiated from aplastic or hypoplastic anemia by the presence of hyperplastic bone marrow. Splenectomy, though not removing the cause may improve the clinical condition and prolong life. HYMAN I. GOLDSTEIN

BOOK REVIEWS

PATHOLOGY. W. A. D. Anderson, M.D., F.A.C.P. 1453 pages, 1183 illustrations, 10 color plates. C. V. Mosby Co., St. Louis, Mo., 1948. Price $15.00.

The list of contributors to this volume reads like "Who's Who", and they add a great deal to the excellent text.

Dr. Anderson states that in this volume he has brought together the specialized knowledge of all fields of pathology. The reviewer finds that the subjects described are clear and explanatory, the illustrations are excellent and well reproduced.

There are forty-six chapters, each one complete. To illustrate, the reviewer calls attention to chapter six, page 128, which deals with physical agents in the causation of disease. Here the physician will find a description of the agents, their effect, etc. on the human organism. On page 192, atomic bomb injuries and their effect are described.

The effect of radiation on the gastrointestinal tract is interesting and informative and should be read by the radiologist.

Chapter 15 deals with Rickettsial and Viral Diseases.

Chapter 19 Neoplasma—here the reader will find information which is not found in most books on pathology. The illustrations enhance the text.

On page 783, the lips, tongue, teeth and neck are fully discussed.

Opposite page 812 there are six beautiful color illustrations showing pathologic states of the esophagus. The varices of the esophagus in a case of liver cirrhosis demonstrate the possible source of hemorrhage.

Ulcer of the stomach and duodenum as well as various intestinal lesions—colitis, dysentery, appendicitis, are discussed fully. The various parasitic infestations are discussed and illustrated.

On page 861, the liver, its functions, diseases, etc. are presented in a concise manner. Easy to read, easy to understand and to assimilate.

It would take reams of paper to describe the many other chapters, however, the reviewer wishes to conclude with Chapter 46, dealing with heredity and constitution in disease. It is rather a short chapter but worthwhile to be read.

All in all Dr. Anderson's Pathology should be on the shelf of every physician who is interested in learning more about the human constitution.

OPERATIVE SURGERY. Frederick C. Hill, B.A., M.S., M.D., Associate Professor of Surgery, The Creighton University, School of Medicine, Omaha, Nebraska. Foreword by Charles W. Mayo, M.D. 698 pages, Oxford Univ. Press, 1949. Price $12.75.

This single volume work on general surgery successfully accomplishes the purposes and intentions of the author, an experienced surgeon and teacher. The author has designed and written this book to be an aid to the interne, resident, and young surgeon, who is as yet not experienced. It is not a complete text-book on general surgery. It does not contain important references to the literature. Operations are briefly, but clearly and adequately described by the author.

Highly specialized operations and new technical procedures not yet in common use are omitted from this volume. Adequate comments are made concerning pre- and postoperative care and management. Sutures, ligatures, and instruments, are discussed.

Gallbladder and gastrointestinal surgery are informatively presented.

Gynecological, urological, bone and joint surgery receive careful consideration. Brief discussions are included by the author on breast and thoracic surgical procedures. There are included very brief sections on vagotomy, and surgery of the heart, pleura and lungs, and esophagus (53 pages).

This compact authoritative volume is recommended by the reviewer for daily use by the surgical interne, resident, and young surgeon and for all young physicians and students interested in surgery.

PROGRESS IN CLINICAL MEDICINE BY VARIOUS AUTHORS. Edited by Raymond Daley, M.A., M.D., M.R.C.P. and Henry G. Miller, M.D., M.R.C.P., D. P. M., 22 text figures and 15 plates, 356 pages, Grune & Stratton, New York, N. Y., 1948. Price $6.00.

This book is written by physicians with special experience in their various branches of medicine, and there are presented some of the major clinical developments of the past few years. Some laboratory and experimental discoveries are included, and their uses and limitations are discussed.

Among subjects discussed are the control of infections by the use of antibiotics, sulfa drugs, sources and modes of infection and immunization and, venereal diseases; a special section on tropical medicine covering malaria, kala-agar, amebic dysentery, bacillary dysentery, filariasis, schistosomiasis, yellow fever, vaccination, and smallpox.

There are interesting chapters on gastrointestinal disorders, liver diseases and metabolic disorders. Dr. F. Avery Jones consulting gastroenterologist, British Postgraduate Medical School, presents

discussions on dysphagia, (achalasia, Plummer-Vinson syndrome, esophageal peptic ulcer, achalasia, hiatus hernia) dyspepsia, (gastroscopy, and gastric and duodenal ulcer, hematemesis, perforation), chronic diarrhea, ulcerative colitis, regional ileitis. Infective hepatitis, homologous serum jaundice and liver function tests are given brief consideration.

Dr. George A. Smart, of the University of Bristol, writes on diabetes mellitus, sprue, gout and vitamins.

There are chapters on cardiovascular diseases by Dr. Raymond Daley. The treatment of subacute bacterial endocarditis with penicillin, surgery of congenital heart disease, essential hypertension, angina pectoris, circulatory failure, arteritis, and Kussmaul and Maier's disease, temporal arteritis or Horton's syndrome, disseminated lupus erythematosis and Libman-Sacks disease receive brief, but satisfactory consideration.

Renal diseases are well presented by Dr. Christopher Hardwick in Chapter VII.

Israels, able investigator and research assistant of the University of Manchester, is author of the section on "Diseases of the Blood". Joseph Smart of the London Chest Hospital discusses diseases of the chest, commenting particularly, on pulmonary tuberculosis, bronchiectasis, abscess, malignant neoplasma, silicosis, and Boeck's sarcoidosis. Diseases of the nervous system, including discussions on Sciatica, electroencephalography, poliomyelitis, myasthenia gravis, and prefrontal leucotomy are briefly presented by Henry G. Miller, neuropsychiatric specialist. Dr. Miller, also, is author of the Chapter on "Psychosomatic Medicine" and, of course, discusses peptic ulcer, mucous colitis, anorexia nervosa, and certain disturbances of the cardiovascular system.

There is a concluding chapter on "The Chronic Rheumatic Joint Diseases" by Philip Elman, of the St. John Clinic for Rheumatism.

This volume contains informative material of interest and value to many clinicians, internists, and general practitioners. It is recommended as an up-to-date book on the progress in clinical medicine.

SOME COMMON PSYCHOSOMATIC MANIFESTATIONS. J. Barrie Murray, M.A., M.D., M.R.C.P., Diagnostic Physician, Travistock Clinic, Honorary Physician, Bolingbroke Hospital, Honorary Physician, The Margaret Street Hospital for Diseases of the Chest, London. 101 pages, Oxford University Press, N. Y., 1949. Price $2.50.

The author believes that sixty per cent of cases seen in the medical out-patient clinic are psychiatric in nature.

The general practitioner "should be best able to recognize the psychiatric aspect of his cases and by instinct or by experience, should be able to treat this aspect".

Darwin (1872) made observations of the expressions and movements of animals under the influence of various emotions. Similar reactions occur in human beings, with more complex and variable manifestations formed and influenced by the environment and previous upsets or experiences in the life of the patient. Emotion is thus expressed as a physical symptom.

This publication was prompted by many years of experience in the clinical examination of psychoneurotic patients. The author discusses satisfactorily and adequately, effort syndrome, its symptoms, physical signs, and differential diagnosis, and "the low-back syndrome". The treatment of patients complaining of psychosomatic manifestations by psychotherapy is emphasized. T. Lewis is quoted by the author, concerning patients complaining of precordial pain, referring to graduated exercises and "mass psychotherapy". The author feels that sexual impotence is a common symptom in psychiatric disorders, and is usually temporary during phases of psychic upset, disappearing with improvement of the psychiatric condition. There is very rarely only, a physical cause of sexual impotence, and any successful treatment can be carried out by the psychiatrist. Physical therapy is unnecessary. "Effort Syndrome" is ill-named because the symptoms may occur at rest, and not on exertion! Powell and Osler treated the condition by suggestion. A. Eulenberg speaks of "pseudo-angina vasomotoria" (1878), and it is identical with hysterical angina. The author thinks these names are preferable to the term "effort syndrome"—because this latter name has an adverse effect on patients who are already in a highly suggestible psychological state. This little monograph does not contribute anything new nor will it be helpful to the experienced internist or clinician or psychiatrist. It is, however, a work that can be profitably read by the medical student, the nurse, and general practitioner and is recommended by the reviewer as a practical little book for such readers.

THE 1948 YEAR BOOK OF ENDOCRINOLOGY, METABOLISM AND NUTRITION: Endocrinology edited by W. O. Thompson, M.D., Clinical Professor of Medicine, University of Illinois College of Medicine, Attending Physician, Henrotin Hospital, and Grant Hospital of Chicago. Metabolism and Nutrition edited by Tom D. Spies, M.D., Chairman Department of Nutrition and Metabolism, Northwestern University, School of Medicine, Director, Nutrition Clinic, Hillman Hospital, Birmingham, Alabama. 544 pages. The Year Book Publishers, Inc., Chicago, 1949. Price $4.50.

This compact little volume edited by Doctors Thompson and Spies, contains many interesting abstracts of informative and worthwhile articles that have appeared in many of the leading journals. The book is highly recommended for all types of readers—medical students, clinicians, and general practitioners—as an up-to-date "refresher" course in the literature of 1947-1948.

DIABETES AND ITS TREATMENT. Joseph H. Barach, M.D., F.A.C.P., Associate Professor of Medicine, University of Pittsburgh, Senior Medical Staff, Presbyterian Hospital, Medical Director, Outpatient Department of the Medical Center Hospitals, School of Medicine, University of Pittsburgh. 326 pages text and 189 pages of diets and recipes. Oxford University Press, New York, 1949. Price $10.00.

This newest volume on "Diabetes and Its Treatment" by an experienced specialist in the study and treatment of diabetes brings up-to-date our present knowledge of this important subject.

The first chapter gives an interestingly informative "History of the Disease" beginning with Papyrus Ebers (Ca. 1500 B.C.) and reference to abnormal polyuria (diabetes?), Celsus' (30 B.C.-27 AD?) good description of diabetes, Aretaeus of Cappadocia (30 A.D.-90 A.D.?) who gave us the term "Diabetes" (to pass through), Galen (131-201 A.D.), Rhazes, Aircenna, Sylvanus (1478-1555), Paracelsus, Cardan (1501-1576), and Willis (1621-1675) who claimed diabetes was primarily a disease of the blood, the sweetness of diabetic urine, and differentiated diabetes mellitus from diabetes insipidus. Reference is made to the work of Ettmuller in 1683, Latham (1811), Bouchardat (1806-1886), Claude Bernard, Pavy (1829-1911), Von Moorden, Naunyn, who first employed the term "acidosis", Opie, Woodyat, Allen, Joslin, Banting and Best, Hagedorn, John Abel, and finally the work on "Alloxan Diabetes" by Dunn, Sheehan, and McLetchie in 1943.

The author gives a description of the diseases and its background, discusses etiological factors, clinical diagnosis, laboratory diagnosis, clinical pathology, complications and their treatment, tuberculosis and diabetes guides in the treatment of children with diabetes.

Prevention and prognosis, treatment, the use of insulin and its effects, are all satisfactorily and adequately covered. In addition to the 326 pages of text, there is a section with 189 unnumbered pages giving a detailed list of men's diets, women's diets, children's diets, luncheons, and emergency diets, and recipes. There is an index of ten pages. With the exception of the excellent bibliography following the first chapter (historical) there is a scarcity of bibliographic references. The 189 pages of diet lists should be numbered.

This 1949 volume on "Diabetes" is recommended for use as a desk copy by all internes, residents, and general practitioners, and by all physicians treating diabetic patients.

Dietitians should find the 189 pages on "diets" for men, women and children, of great help.

HUMAN PHYSIOLOGY. A PRACTICAL COURSE. C. G. Douglas, C.M.G., M.C., D.M., F.R.S., Professor of General Metabolism and Fellow of St. Johns College, Oxford and the late J. G. Priestley, M.C., D.M., Reader in Clinical Physiology, University of Oxford. 258 pages. Clarendon Press, 1948. Price $5.50.

Dr. Priestly died in 1941. Dr. Douglas in preparing this third edition had the assistance of Dr. F. C. Courtice. This little volume should be of considerable interest and help to the students of medicine, and to all physicians, interested in the physiology of respiration, respiratory exchange and energy production, blood, gases of the blood, circulation, various kidney tests, and the physiology of the alimentary canal.

The Chapter (III, IV and V) covering investigation of the blood, the gases of the blood, and the circulation (41 pages) are of particular interest, and include valuable informative material.

This book is recommended for the student, as well as for the general practitioner.

YOUNG-MILLER'S HANDBOOK OF ANATOMY. Revised by George W. Miller, M.D., F.A.C.S., Professor of Anatomy, Temple University Dental School, Philadelphia, Surgeon, Norristown State Hospital, Norristown, Pa. Ninth edition, 493 pages, 142 illustrations, 4 in color. F. A. Davis Company, Philadelphia, 1948. Price $5.50.

This book has now reached its ninth edition which bespeaks its usefulness through these many years to students and physicians alike. This book is recommended for medical and dental students, and for nurses, and art students.

ACUTE INTESTINAL OBSTRUCTION. Rodney Smith, M.S., F.R.C.S., Assistant Surgeon, St. George's Hospital, London, Consulting Surgeon, Wimbledon Hospital, Hunterian Professor, Royal College of Surgeons, with a Chapter on Radiological Diagnosis by Eric Samuel, M.D., F.R.C.S., F.F.R., D.M.R.E., Late Radiologist, The Middlesex Hospital, London, foreword by Rupert Vaughan Hudson, F.R.C.S., 259 pages, Williams and Wilkins Company, Baltimore, 1948. Price $5.00.

This small volume brings up-to-date our present knowledge on the subject of acute intestinal obstruction.

There is an interesting historical chapter giving the history of surgery of acute intestinal obstruction. The author is in error in stating that "it was not until 1836 that the first successful resection of gut was performed by Dieffenbach". The cases reported by John Needham (1755), Ramdohr (1727), Charles A. Luzenberg (1836) of New Orleans, are overlooked by the author. No mention is made of the experiments on dogs in 1805, 1841, and 1842 by Dr. Thomas Smith, of St. Croix! Of course, others, like Kocher, Koeberle, Hagedorn, Czerny, Nicoladoni, Molodenkow and Minn, Roggenbau, and Byrd, of Illinois, performed successful gut resections. All these early operators, the author fails to mention.

The author adequately but briefly, discusses simple occlusion of the intestine, high and low small gut occlusion, large gut occlusion, closed loop obstruction, strangulation, functional obstruction, protein deficiency in intestinal obstructions; clinical diagnosis, roentgenography and treatment receive consideration, as do chemotherapy, suction drainage and intubation (Miller-Abbott tube), preoperative management, and operative technic, imperforate anus, developmental bands, strangulated inguinal and femoral herniae, hernia through the mesentery, obstruction by bands and adhesions, volvulus of the colon, idiopathic intussusception, strictures, and mesenteric vascular occlusions are concisely discussed. There is a bibliography of 350 references.

This little monograph on acute intestinal obstruction is recommended to medical students, internes and residents and to surgeons and clinicians.

LIVING ANATOMY. A PHOTOGRAPHIC ATLAS OF MUSCLES IN ACTION AND SURFACE CONTOURS. Professor R. D. Lockhart, M.D., Ch.M., Regius Professor of Anatomy, University of Aberdeen. 71 pages, 148 figures. Oxford University Press, N. Y., 1948. Price $4.00.

This book illustrates beautifully muscle action in the living man and woman. The many illustrations are excellent. This monograph is recommended to artists, sculptors, medical students, and those interested in physical culture and instruction.

The photography by Alexander Cain, A.R.P.S. is excellent, and deserves special mention.

CLINICAL NEUROLOGY. Bernard J. Alpers, M.D., Professor of Neurology, Jefferson Medical College, Philadelphia, Neurologist to the Jefferson, Pennsylvania and Wills Hospitals, Philadelphia. 797 pages. F.A. Davis Co., Philadelphia, 1947. Price $8.00.

This textbook on "Clinical Neurology" is highly recommended for medical students, residents and internes, and general practitioners of medicine.

All the usual, and many of the unusual neurological conditions encountered in practice are adequately described by an experienced teacher of neurology.

Examination of the nervous system and interpretation of neurological symptoms and signs as well as the topical diagnosis of nervous disease are clearly and instructively presented. Headache, vertigo, coma, pain are discussed in Chapter IV. Disease of the Spinal Cord, diseases of the Meninges, Encephalitis, syphilis of the brain, vascular disease of the brain, and tumors of the brain are well presented.

The author also discusses abscess of the brain, cranial trauma, epilepsy and convulsive states, extrapyramidal diseases, multiple sclerosis, intoxications and injuries by physical agents, vegetative nervous system and endocrine disorders, nuclear amyotrophies and myopathies, degenerative diseases, congenital and developmental disorders, and diseases of the skull and vetebrae. All medical libraries in hospitals and medical schools should have this volume readily available for their students and internes.

THE EARLY DIAGNOSIS OF THE ACUTE ABDOMEN. Zachary Cope, B.A., M.D., M.S., F.R.C.S., Surgeon to St. Mary's Hospital, Paddington, Senior Surgeon to the Bolingbroke Hospital, Wandsworth Common, Late Hunterian Professor, and Arris and Gale Lecturer, Royal College of Surgeons. Ninth edition. 262 pages. Oxford University Press, New York, 1948. Price $4.25

The ninth edition of this little volume written in 1945, now appears in its third impression in 1948. This book has been improved with some alterations and corrections and some additions. There are 38 figures giving the reader informative illustrations. There are interestingly instructive chapters on diagnosis, appendicitis, perforation of gastric or duodenal ulcer, acute pancreatitis, acute intestinal obstruction, intussusception, cancer of the large bowel, volvulus, strangulated and obstructed heniae, ectopic gestation, cholecystitis, the colics, abdominal injuries, hepatitis, dysenteric perforation and typhilitis, liver abscess, typhoidal ulcer perforation are briefly considered in Chapter XVI (7 pages) and acute peritonitis is briefly discussed in Chapter XVIII (10 pages).

Renal colic, pyonephrosis, and abnormalities of urine are mentioned in "acute abdominal disease with genitourinary symptoms".

A closing chapter (XIX—of 13 pages) adequately and concisely presents "Diseases which may Simulate the Acute Abdomen"—influenza, diabetic coma (impending), typhoid fever, tuberculous peritonitis, food poisoning, acholuric jaundice, thoracic diseases—pleurisy or pleuropneumonia, acute cardiac disease pericarditis endocarditis, angina pectoris, diseases of the spinal cord, acute osteomelitis of the dorsal or lumbar vertebrae, retroperitoneal effusions, dissecting aneurysm, and pelvic subperitoneal infections. This is a worthwhile informative little volume on the early diagnosis of the acute abdomen.

FOR DOCTORS ONLY. Francis Leo Golden, foreword by William E. Mountford, M.D., illustrated by Barye Phillips, 273 pages, Frederick Fell, Inc., New York, 1949. Price $2.95.

This is a "humorous" book.

It consists of a compilation of the funny experiences of medical practice and the allied professions well presented. This book will bring laughs from patients, nurses, medical students, and doctors. A good book to have on the waiting room table.

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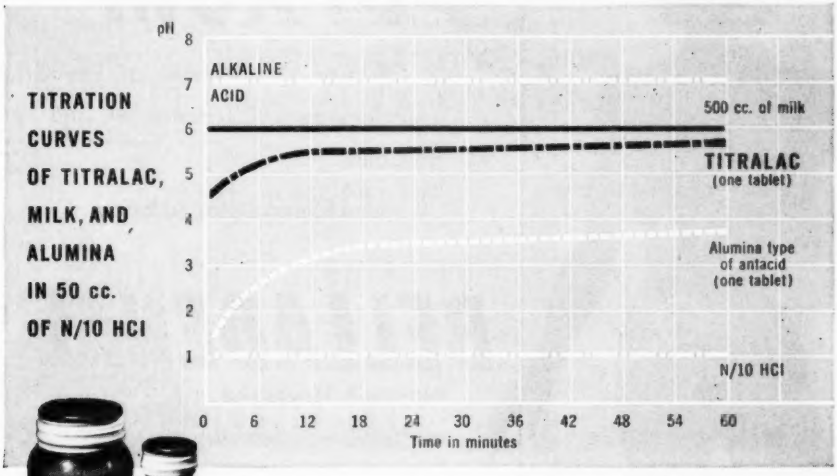

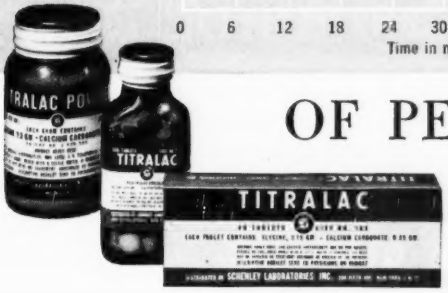

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REFERENCES

1. Rossett, N. E., and Flexner, J.: Ann. Int. Med. 18: 193 (1944). 2. Freezer, C. R. E.; Gibson, C. S., and Matthews, E.: Guy's Hosp. Reports 78: 191 (1928). 3. Aaron, A. H.; Lipp, W. F., and Milch, E.: J. A. M. A. 139: 514 (Feb. 19) 1949. 4. Kirsner, J. B., and Palmer, W. L.: Illinois M. J. 94: 357 (Dec.) 1948. 5. Kimball, S.: in Practice of Medicine (Tice). Hagerstown, Md., W. F. Prior Company, Inc., 1948; p. 210. 6. Special Article: M. Times 76: 10 (Jan.) 1948.

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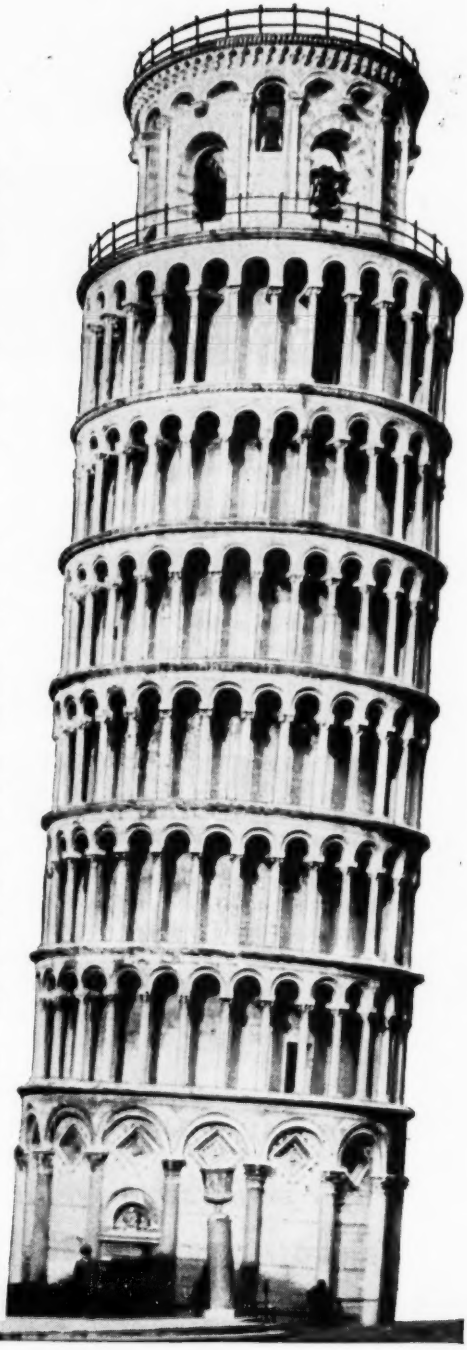

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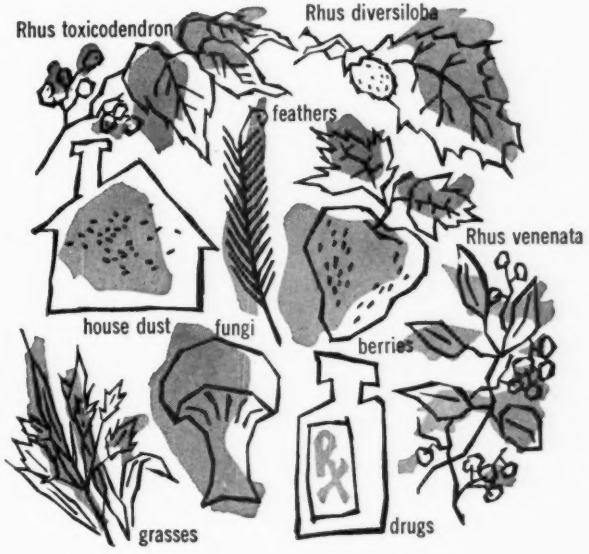

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